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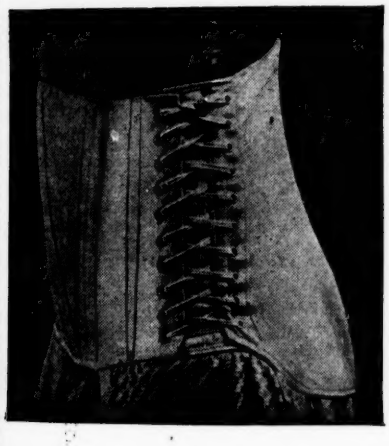

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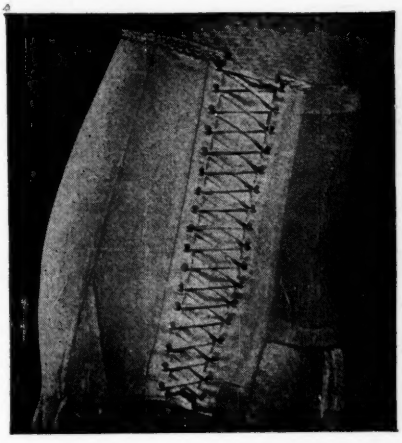

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A CLINICAL STUDY OF CHRONIC ULCERATIVE COLITIS*

BY EDWARD S. EMERY, JR., M.D.,† AND PAUL H. WOSIKA, M.D.†

EXPERIENCE in caring for patients with ulcerative colitis is a necessary qualification of providing them with the best kind of treatment, because the disease entails many diverse complications or sequelae and because no specific form of therapy has been developed. Hence most physicians are handicapped in their treatment of the occasional case; for the disease is just uncommon enough to prevent them from acquiring any extensive experience with it. Its comparative rarity is shown by the fact that only seventy-three patients with ulcerative colitis entered the medical wards of the Peter Bent Brigham Hospital out of 27,659 admissions in twenty years. Therefore we hope that a report of these cases may be of some value to those who have had less opportunity to treat this disease.

DIAGNOSIS

The diagnosis of ulcerative colitis is made by the finding of blood and pus in the stools. As the disease varies greatly in the severity of its manifestations, patients may be observed with little or no diarrhea, in fact one of our patients had formed movements throughout the course of her disease. Other conditions may give blood and pus. But the finding of pus, particularly if associated with blood, should always suggest the possibility of ulcerative colitis. The presence of these substances, in what otherwise would be considered a case of ordinary diarrhea, practically assures the diagnosis. Whenever possible it is wise to have an x-ray and a proctoscopic examination, and these should always be done where the presence of a true diarrhea is doubted. X-rays are useful in ruling out such conditions as new growths and diverticula. Sigmoidoscopic examinations permit visual confirmation of the fact that the blood and pus come from an ulcerative mucous membrane in the large majority of the patients in whom the process extends to within reach of the instrument.

ETIOLOGY

After the presence of an ulcerative colitis is suspected, it is important to determine the underlying cause which may influence the prognosis and treatment. Tuberculosis usually does not present the full-blown picture of ulcerative colitis except in patients who have an advanced tuberculous process in the lungs. However, this is not always so, as is illustrated by one patient in whom tubercle bacilli were later obtained from the walls of the ileum after an operation for a supposed "idiopathic ulcerative colitis". A positive diagnosis of tuberculous enteritis has been made in thirty-nine patients at the Brigham and a questionable diagnosis in nineteen others. Its incidence is therefore high enough to warrant its consideration whenever a chronic diarrhea is encountered. It most commonly involves the ileocecal region, resulting in a mild to moderate diarrhea. Unless quite extensive, the presence of blood and pus is frequently missed, and the tubercle bacillus is only rarely found in the stool. Proctoscopic examination is usually of little avail, and one must rely on the x-ray to detect its presence. Brown and Sampson[1] have shown the value of the latter in the presence of pulmonary tuberculosis, but in its absence an x-ray interpretation alone must be accepted with caution[2]. Confusion may occur with carcinoma and the so-called "regional ileitis". The demonstration of an irritable focus in the ileocecal region, associated with tuberculosis of the lungs, warrants a diagnosis of tuberculous enteritis in those with diarrhea for whom there is no other adequate explanation. With the absence of pulmonary involvement, such a lesion in the ileum is more probably in the nature of a "regional ileitis".

Infection with the *Entameba histolytica* gives a clinical picture quite similar to the idiopathic type of ulcerative colitis and should always be suspected in these cases. The recent Chicago epidemic has brought to the attention of the profession the widespread incidence of the parasite throughout the United States, a fact which has not apparently been generally recognized hitherto[3]. Fifteen cases of amebic dysentery have entered the Peter Bent Brigham Hospital since its opening in 1913. Although several of these patients were seen shortly after the war, the others were observed at scattered intervals during the last twenty years. Only one of these patients received his infection in New England, the rest having brought theirs from tropical countries, but the recent literature suggests that it is rapidly becoming endemic in the United

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States. The diagnosis can be definitely made only by the recognition of the organisms in the stool. This is not difficult with a little practice. The usual method is to search for the parasite in the scrapings from the base of an ulcer. For those who do not use a proctoscope, a fairly satisfactory method is to insert a rectal tube just after a movement and have the patient strain. The organisms are frequently found in the mucus that is withdrawn with the tube. Whenever the organisms are not found, but their presence is still suspected, a therapeutic test with subcutaneous injections of emetine hydrochloride is justifiable and often helpful. This drug usually will alleviate the symptoms of amebic dysentery fairly promptly; whereas it may increase a diarrhea due to other causes. It seems doubtful if the complement fixation test is of any more value than microscopic studies of the stools[4].

Bacillary dysentery due to the Shiga and Flexner organisms is another cause of ulcerative colitis. During acute attacks one can make the diagnosis by direct cultural methods, using a litmus agar plate. Later, when the disease has become chronic, it is usually impossible to make a definite diagnosis. The organisms cannot be cultured and the agglutination reactions and attempts at therapy by serum and vaccines have yielded equivocal results[5]. It is probable, therefore, that a certain number of patients with ulcerative colitis started with a bacillary dysentery, a view concurred in by Hurst[6].

After the above etiologic agents have been ruled out, a large group of individuals are left in whom a diagnosis of "idiopathic ulcerative colitis" is made because no cause for the condition is found.

The condition has been ascribed to various agents perhaps the more important ones being a deficiency in vitamines, a neurogenic disturbance and some bacterial agent. However, the evidence does not convincingly incriminate any one of these suggestions, and the reader is referred to the recent article of Paulson[7] for a discussion of the present status of the various etiologic agents. As a matter of fact, the disease behaves so differently in different patients as to suggest that all cases of idiopathic ulcerative colitis are not due to a single etiologic agent, although all cases seem to be complicated by, if not the result of, infection which explains most satisfactorily the fever, leucocytosis, and later scarring of the tissues. At present the physician is obliged to treat the patient on the theory that the real cause has not yet been discovered. Theoretical considerations are of no value, and treatment must be based on the results of experience.

There are seventy-three patients with idiopathic ulcerative colitis in the series. Thirty-five were males and thirty-eight were females. Although the disease is somewhat prone to attack younger persons, the age at onset varied from ten to sixty years with an average age of thirty. The duration of symptoms before entering the hospital varied from one week to twenty years.

BEHAVIOR OF THE DISEASE

The most striking feature of the disease called idiopathic ulcerative colitis is the marked variability in its onset and course. The onset in thirty-five of the seventy-three cases was mild. These patients noted some increase in the number of movements which were somewhat softer than normal. Some experienced slight discomfort in the lower abdomen or rectum and a few had noticed traces of bright blood. In twenty-one patients the onset could be characterized as moderate. Some of these patients complained of the same symptoms as those listed under a "mild" onset, the only difference being that the symptoms were somewhat more exaggerated. Others noted a definite diarrhea and a moderate loss of blood. Some of the keener observers recognized the presence of some pus. The disease started in seventeen patients in a definitely severe form. These patients developed without warning an intense diarrhea with ten to twenty evacuations in the twenty-four hours, and one man estimated that his bowels moved one hundred times in the day. Within a few hours the discharges became bloody and large quantities of pus may have been passed.

The subsequent course of the disease varied as much as the onset. It was classified as mild in twenty-seven, moderate in twenty-five, and severe in twenty-one patients. But an important fact which the clinician should keep in mind is that the course cannot be foretold by the type of the onset. The subsequent behavior of the disease in the thirty-five patients with a mild onset was mild in sixteen, moderate in eleven and severe in eight. Of the twenty-one patients with a moderate onset, eight had a mild course, nine had a somewhat moderate course, and four a severe course. Again of the seventeen patients starting with a severe onset, three had a mild course, five a moderate course and nine a severe course. This knowledge of how completely the disease may change in intensity is important to the clinician. A guarded prognosis should be given the patient with mild symptom, whereas, a severe onset does not foretell a fatal termination or even a bad result. Two of the patients with a most severe onset are now well, one having been entirely free from symptoms for seven years and one for five years.

Because the disease may become very severe, one is not justified in permitting a patient with mild symptoms to take inadequate treatment. Once the diagnosis of ulcera-

tive colitis is made, the most careful and intensive treatment should be pushed until every evidence of the trouble has been eradicated, or the conclusion is reached that no further improvement can be expected. One of the most striking examples of this occurred in a girl thirteen years old. When first seen she complained of mild abdominal sensations. The only positive finding was the appearance of the intestinal mucous membrane through the sigmoidoscope. It showed the very mildest type of change seen in cases with a colitis. The membrane had lost the normal velvety appearing surface and looked somewhat rough and granular. For the next two months there was no diarrhea or any blood or pus in the stools. Yet without warning, she developed an acute bloody dysentery which progressed rapidly to a fatal termination within three weeks.

The disease must be considered a chronic one although many relapses result from a failure to continue treatment long enough. This will be considered later under the head of treatment.

Although the disease is difficult to treat, and the prognosis is always uncertain, the results of treatment are not discouraging. The end-result is known in thirty-three patients, of whom ten are well, four are improved, and seven unimproved, but living, and twelve are dead. The best results occurred in those whose treatment was most conscientiously supervised and followed by physician and patient. More adequate treatment for the others would have improved, decidedly, the final figures for the group.

SYMPTOMS

The symptoms are much the same in all cases, the differences being more quantitative than qualitative. They consist of diarrhea, intestinal distress, and the symptoms of infection.

The diarrhea may be mild or severe. One patient as already stated, never had a loose movement throughout the course of the disease. Cases of moderate severity may have four or five movements a day; the severe cases frequently have from ten to thirty movements. The amount of discomfort has little relation to the severity of the diarrhea. Many patients with twenty to twenty-five movements have little or no pain; others with only four or five movements may have considerable discomfort. Rather severe tenesmus occurs when the rectum is involved. Severe pain, particularly if associated with localized tenderness, may signify an impending perforation.

Nearly all patients show some rise of temperature which in the more severe cases may reach 103° F. and give a typical picket fence chart. This has suggested the presence of a septicemia, but blood cultures have been negative in our experience.

The height of the fever usually is in direct proportion to the severity of the local process, as observed by the proctoscope. Yet, a high fever is not always associated with a marked diarrhea.

The white cells rarely increase in number, even in the presence of rather high temperatures. The highest count in the series was 22,000 in a very ill patient who probably suffered from a perforation. The next highest leucocytosis was 17,000. Several patients had a white count of 11,000 to 13,000, but the great majority had counts from 6,000 to 8,000. A high white count suggests involvement of the peritoneal coat and the possibility of a beginning perforation.

The degree of bleeding also varies over a wide range. It rarely is a direct cause of death, but the secondary anemia, which so frequently develops in the severer cases, may be a very troublesome complication of treatment. Sixty per cent of the cases had normal bood counts. The red blood count was five million or higher in 23 per cent, from four to five million in 51.4 per cent, three to four million in 23 per cent and two to three million in 2.8 per cent of the cases.

Other complications which are frequently present and must be kept in mind are dehydration from the severe diarrhea, loss of appetite and parotitis.

TREATMENT

The treatment of idiopathic ulcerative colitis is the treatment of a chronic disease of unknown etiology, involving an infection of the colonic mucous membrane. Therapeutic procedures may, therefore, be divided into those designed toward maintaining or improving the patient's general condition and those aimed toward treatment of the local condition. Physicians must keep clearly in mind into which category each procedure falls, if they are to treat these patients intelligently; for it occasionally happens that a procedure which will improve the general health of the patient may disturb the local condition.

The first duty of a physician, when he sees one of these patients, is to inform some competent member of the family about the nature of the disease, its unknown etiology, chronic course, and the pros and cons of the medical and surgical treatments. Be sure to warn them that there will very probably come a time when every one will be discouraged and may be urging some different kind of treatment. The physician is better able by this means to retain the families' confidence when he needs it most. This is important because it commonly happens that discouragement leads to frenzied attempts to try some new treatment on the theory that anything is better than to continue with something that is not producing results. The problem is the same with this disease as with tuberculosis,

but the laity is not yet educated to accept a similar type of hygienic treatment.

The treatment of the patient seen in the first few days of a sudden and acute attack should be the same as any acute diarrhea. Examination of the stools is sufficient for a diagnosis and x-rays and proctoscopic examinations are contraindicated at this stage because they tend to aggravate the existing pathologic condition. Complete bed rest with a diet limited to boiled milk is indicated together with medication designed to control the diarrhea. This is best administered with large doses of bismuth subcarbonate and adequate amounts of opium. Many of these patients will recover promptly on this régime, and it is important to continue treatment until the condition is adequately healed. One of the greatest mistakes is to allow patients to stop treatment too early, and a failure to treat adequately these patients in the early part of the disease is responsible for many of the chronic cases. The diet may be gradually enlarged and the medication decreased after the bowel movements have become normal. A wise rule to follow in the acute cases is that a patient must remain in bed for a minimum of one week after the stools are normal microscopically and after the patient is on a normal diet.

In the treatment of the chronic case there is one rule of which the physician should be ever mindful and that is attention to details. We know of no condition in which detailed study of the patient and care as to minute points of treatment is more important than in ulcerative colitis.

General care of the patient is most important. Complete bed rest is essential for the less severe forms as well as for the patient that is seriously ill. Mental relaxation is fully as important as physical rest. Most of these patients have very similar and rather characteristic psychological reactions. They are prone to be uncoöperative, rather introspective, often selfish and usually want to have their own way. Their reactions are those of "a spoiled child". Although their psychology frequently is very trying to the physician, it must be treated since the nervous system influences the intestinal tract.

Many of these patients lose large quantities of fluids through the bowel, and dehydration must be anticipated. If an adequate amount of liquids is not taken by mouth, hypodermoclyses of physiological saline should be utilized to prevent drying of the tissues. Although this seems to be an obvious statement, it is surprising how often one sees in consultation a patient whose tongue is parched and who is obviously suffering from marked dehydration.

As all these patients lose some blood, they are always potentially anemic if not actually so. Hence iron is indicated preferably by mouth, but if the gastro-intestinal tract does not tolerate it well, it can be given hypodermically.

It is our custom to expose patients to the ultraviolet rays by means of sunlight or quartz lamps entirely on empirical grounds. The value of these rays in intestinal conditions was originally stressed for tuberculous enteritis[8]. Observers at Saranac have felt that very excellent results are obtained by exposure of the abdomen to this therapy. We have, therefore, applied it to these patients since 1924. Although it has not been possible to demonstrate any positive effect from this therapy, there is reason to feel that it is of some value. As it can do no harm, it seems wise to give these patients the benefit of anything that may be helpful.

Treatment of the diseased part should aim toward supplying rest to the affected organ, a well-established principle of therapy. This procedure, therefore, requires a consideration of diet and drugs. The diet must be such as to produce as little stimulation and irritation to the colon as possible. Eliminate all substances such as cellulose, seeds, gristle, etc., which stimulate mechanically. Foods with no residue but which through some chemical effect increase peristalsis, as the fruit juices, must also be used with caution. Owing to recent investigative studies, many physicians and dietitians have asked about the use of milk. It has been shown[9] that the ileal discharges contain a larger proportion of solid material with a milk diet than with many other foods. But it has not been shown that this type of solid material causes undue stimulation of the colon, and therefore, work of this kind does not disprove the empirical evidence accumulated through years of experience that milk is a suitable food for diarrhea. Its general dietetic value is so great, that it should be included as a worth while part of the diet. The belief of many physicians that meat is contraindicated is not well founded. Beef, lamb and chicken muscle is thoroughly digested and does not disturb the colon. Its use is desirable by increasing the palatability of the diet and by supplying proteins to patients who are losing large quantities in their discharges. We have included a list of foods which will supply an adequate diet, with the exception of the vitamines. These should, therefore, receive consideration. Cream and butter supply sufficient vitamin A. Vitamin B may be given by yeast cakes, or if these prove to be troublesome, by some special preparation. Orange and tomato juice can be used to supply vitamin C. But there is no reason for giving them in large quantities as is sometimes done. The evidence that ulcerative colitis is due to a vitamin deficiency is too slight to offset the tendency of these substances to increase the diarrhea. Only

an amount sufficient to prevent scurvy is necessary, and it is better to begin with daily doses of two tablespoonfuls. If this is well tolerated, the dose may be increased. Vitamin D is easily supplied by ultraviolet light or irradiated ergosterol. Because liver has been shown to contain a substance that is lacking in patients with pernicious anemia, it seems well to include it in the diet with the idea that it may serve as a storehouse for other important substances of which we are still ignorant.

A diet such as this supplies all that is now known to be required for continued good health and is compatible with the principle of giving rest to the diseased colon. Drugs must supply the further rest needed. Bismuth, tannigen and opium are the most useful in our experience. The subcarbonate of bismuth is better than the other compounds as it is as effective and does not introduce the danger of poisoning like the nitrate. A teaspoon of bismuth subcarbonate three to five times in the twenty-four hours is not excessive. Tannigen in the milder cases sometimes works very well and occasionally better than bismuth, so that it is worth trying on cases that do not respond well to bismuth. It may be given in one gram (15 grain) doses, three to five times a day.

Opium is indicated in all the more severe cases and enough should be given to bring the bowels under control. Large doses are frequently required over long periods of time. Physicians often give inadequate doses as they are afraid of developing addiction to the drug, and the treatment is ineffective for this reason. But its use may save a patient that would otherwise die from the effects of a marked and prolonged diarrhea. Since the danger of producing an addiction in these patients is not great, it should be used without question to save life. We have never seen a patient with ulcerative colitis develop addiction even after very prolonged use. Usually they are desirous of stopping it and do so with little annoyance.

It is our custom to give it in the form of the tincture of deodorized opium, in doses of eight to fifteen drops, three to eight times in the twenty-four hours. This is a little pleasanter to take than paregoric. The dose can be regulated more easily than when opium is given in tablet form, and the fact that it is more unpleasant than tablets is useful when it is no longer necessary.

The general treatment outlined so far must be persisted in until the number of stools diminish and the quality of the stools improve. Then the amount of opium may be decreased. Experience helps in determining when to make a change. It may be desirable to decrease the opium for the patient whose stools have decreased from fifteen to twenty a day to five a day, whereas one may wait with the patient who started with five stools until he is having only one. But the point to remember is that changes in treatment must be guided by the condition of the colon as evidenced by the stools. The changes that we look for are a decrease in number, the disappearance of blood and then pus, and then improvement in consistency from watery evacuations to formed movements. Changes in the routine of treatment should take place in the following order, a decrease and finally the elimination of opium, decrease of bismuth and finally an increase in the diet. After the stools are formed, the diet may be increased step by step starting with purées of the simpler vegetables such as spinach, carrots, beets and peas. After these are tolerated in reasonable quantities, cooked pears, peaches, or apricots may be carefully added. But it must be remembered that additions are governed by the response of the intestine. If the stools become softer and the number increases, the intestine is not yet ready for the addition and it must be stopped. Good results will not be obtained in this disease unless meticulous care and attention to detail is used in the treatment.

The exact status of vaccines, particularly the so-called Bargen's vaccine, has not yet been definitely established, although the reports from the Mayo Clinic suggest that better results are obtained with its use than without it. We have tried it on a number of patients without being able to decide its value. The three patients who have taken it most conscientiously are well to-day, and all three were severe cases when the injections were started. But it is difficult to say to what degree the good results are dependent upon the vaccine and how much upon the patients' careful attention to all other directions about treatment. A word of caution should be given in the use of these vaccines, however. Care should be taken to use small enough doses to prevent a general reaction which often results in an intensification of the colonic process. We have had no experience with Bargen's serum in the treatment of these cases.

Colonic irrigations continue to hold the attention of the laity and the profession for the same reasons that the ancients believed that the intestines contain material detrimental to the health of the individual. In spite of the fact that no one has yet shown at what point in the tract the intestinal contents cease to be an asset and become a liability, in spite of the fact that anyone can demonstrate that irrigations definitely result in irritation of the colon, physicians continue to prescribe them and patients to demand them. The urge to use them in ulcerative colitis is strengthened by the presence of an obvious infection, although the possibility is so slight of being able to overcome infec-

tion in the intestines which normally teem with bacteria. Physicians also lose sight of the fact that their use also offsets all the procedures that are being advocated to supply the cardinal principle of rest to the affected part. We wish to state that we have never seen any improvement result from enemata, medicated or otherwise in ulcerative colitis and have frequently seen harm ensue. All the more common varieties of enemata have been tried on the patients in this series without benefit. These include enemata of olive oil, silver nitrate, starch, zinc sulphate, quinine, boric acid, mercurochrome, potassium permanganate, acidophilous organisms, gentian violet and irrigations through the appendix. Irrigations in our experience are ill-advised and should not be used.

The question of when to use surgery is not always easy to decide. Although the indications for its use are better understood than formerly, one still encounters physicians who argue that surgery is a better form of therapy than medicine. But experience has shown that surgery is not a cure. If it were, there would be no question about using it in all cases as medical treatment is admittedly unsatisfactory and uncertain. The objection to surgery is that the disease continues to be active, scarring occurs with consequent shrinkage, and there is little hope of ever reuniting the intestine. Let the reader who disagrees with this statement recall how many cases he has seen in which the operation could be undone.

There are two, possibly three, indications for surgery.

1. When a sufficient amount of scarring has developed to preclude all hope of cure.
2. When perforation threatens or occurs.

It is often difficult to resist the urge to operate in acute cases which are going from bad to worse on medical treatment, on the theory that anything is better than allowing the patient to die "without doing anything". Friends and relatives implore a change, and the family physician, or at times another consultant, suggests surgery as a last hope. However, we have seen patients who are apparently at death's door suddenly rally and get well. It is these cases which make one skeptical as to whether the improvement of some cases after surgery are merely coincidental, and the same would have happened if thorough medical treatment had been continued. Moreover, one hesitates to add the burden of surgery to a patient already at a low ebb.

The situation is different with the chronic cases. These patients may continue to bleed, lose weight, and their condition appear serious, yet they stand an operation very well and immediately thereafter begin to improve. Surgery should be attempted in these cases even though the condition of the patient seems quite precarious. Surgery is also indicated for the chronic patient in whom scarring prevents hope of improvement and who is bothered by the frequent rectal discharges or a continued anemia.

The dangers of perforation are too serious to be trifled with, and surgery should be used whenever there is evidence of impending perforation. The warning signs are severe pain with discrete tenderness, particularly if the tenderness is localized to one part of the abdomen.

Another troublesome complication is loss of appetite. This sometimes follows too much opium. It may also occur when the disease involves the cecal region sufficiently to interfere with the normal intestinal gradient. When anorexia becomes a problem in management, the effect of decreasing the amount of opium should be tried, even at the risk of having the diarrhea increase. If this fails to correct the situation, surgery should be seriously considered. We have seen patients who were unable to eat before an ileostomy, develop a ravenous appetite within twenty-four hours after the operation. Loss of appetite may therefore be listed as another indication for surgery.

Anemia frequently is a serious complication in the treatment of the disease. Patients may lose blood so rapidly in the acute phases that the anemia can be overcome only by transfusions. The chronic cases may also develop such a severe grade of anemia, in spite of large doses of iron, as to become an important factor in the treatment of the patient. Many of these patients improve enormously following transfusions. However, general reactions may result in such a marked disturbance to the colon that the patient is worse off than before. Although we have seen some very excellent results follow transfusion, we have seen enough dire effects to make us very cautious about advising it. One patient was transfused against our advice because of a hemoglobin of 60 per cent, although the red count was 4,000,000, and the patient's stools had very definitely improved. There was a marked recrudescence of the disease with increased diarrhea and a return of bloody stools and it required six weeks before the patient regained the condition she was in before the transfusion was given. It was virtually impossible to overcome the anemia of another patient because the increased bleeding following each transfusion offset the effect of introducing the blood. The decision to transfuse these patients should, therefore, be arrived at only after due consideration, realizing that it is by no means an innocuous procedure.

SUMMARY

The diagnosis and treatment of chronic idiopathic ulcerative colitis are discussed from ob-

servations made on seventy-three cases of the disease. Although the etiology is not known, good results can be expected from adequate nonspecific therapy. An unsatisfactory response to treatment frequently is the result of insufficient attention to the details of treatment. Many relapses occur because the patient is discharged before there is complete restoration of intestinal function, as revealed by examination of the stools. Surgical treatment is indicated for a badly scarred intestine, for the symptoms and signs of perforation, and occasionally for loss of appetite. Otherwise, medical treatment is the best form of therapy.

FOODS THAT MAY BE USED IN DIET FOR ULCERATIVE COLITIS

White bread	Roast lamb	
Crackers, plain	Lamb chops	
Butter	Cream of Wheat	
Milk	Boiled rice	
Cream	Macaroni	
Eggs	Custard	
White meat of chicken	Tea	Salt
Roast beef	Coffee	Pepper
Steak	Fish	Sugar

Any combination of those foods may also be taken.

Examples:

Creamed fish with egg sauce or baked with buttered bread crumbs.
Creamed chicken.
Creamed eggs.
Plain baked macaroni or spaghetti with buttered bread crumbs.

Desserts as:

Ice cream, coffee, vanilla or caramel.
Rice pudding without raisins.
Bread pudding without raisins.
Junket.
Plain blanc mange without fruit.
Bavarian cream without fruit.
Ivory jelly.
Coffee gelatin or coffee sponge.
Spanish cream.
Floating Island.

Vanilla, caramel or coffee only, used for flavoring.

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DARTMOUTH COLLEGE NEWS

Dr. Alfred A. Bielschowsky, eminent German ophthalmologist, arrived in Hanover, November 7, 1934, from Breslau to begin six months of special research work in the Department of Research in Physiological Optics of the Dartmouth Medical School. Dr. Bielschowsky, an authority on motor anomalies of the eyes and until recently professor of ophthalmology and Chief of the Eye Clinic at the University of Breslau, will work with the Department of Research in Physiological Optics on the analysis of abnormalities of vision.

The Department of Research in Physiological Optics at the Dartmouth Medical School has attracted world-wide attention for its work during the past ten years and particularly for its announcement last April of a hitherto unknown eye defect, named aniseikonia. The Department has perfected the Ophthalmo-Eikonometer, an instrument for determining and measuring aniseikonia, and has coöperated with the American Optical Company in developing iseikonic lenses for the correction of this eye defect. In connection with its research work, the Department conducts the Eye Section of the Hitchcock Clinic.

Dr. Bielschowsky, whose arrival attaches a new importance to the Department, has had a long career in ophthalmological work at the Universities of Leipzig, Marburg and Breslau. After obtaining his medical degree at the University of Berlin, he turned to the field of eye diseases and studied in the clinic of Prof. H. Sattler in Leipzig. He was also a research student under Ewald Hering, master of the physiology of space perception, and with F. B. Hofmann, Hering's assistant, collaborated on the study of the fusion movements of the eyes, resulting in the treatment of ocular palsies. Dr. Bielschowsky also did important work on compensatory head postures and squinting, his studies of the latter defect winning him the position of Docent in Ophthalmology at the University of Leipzig in 1900.

He was advanced to University Lecturer in 1906, and from 1907 to 1912 he was head physician at the Leipzig Eye Clinic. In 1912 he was called to the University of Marburg to be Professor of Ophthalmology and Chief of the Eye Clinic, and in 1923 he assumed the same positions at the University of Breslau. Dr. Bielschowsky resigned his Breslau position in September, and will now work at Dartmouth for the next six months.

Dr. Bielschowsky has lectured before numerous ophthalmological societies in this country, principally on the subject of motor anomalies of the eyes, in which field he is a recognized authority and in which his treatise is considered as a definitive work. During the World War, Dr. Bielschowsky founded in Marburg the Educational Institute for Blind Academicians, the first and only institute in which intelligent and capable blind people are helped to pass higher schools and to follow academic studies which will enable them to become teachers or enter the professions. The institute contains a library of 20,000 volumes of scientific literature in Braille.

THE NEW HAMPSHIRE MEDICAL SOCIETY

PERNICIOUS ANEMIA*

BY WILLIAM P. MURPHY, M.D.†

Mr. Chairman, Ladies and Gentlemen:

ALTHOUGH the original liver methods of treating pernicious anemia proved to be satisfactory under most circumstances, it seems to me that the introduction and development of a method of giving the extract parenterally has been a definite step in advance in the determination of adequate treatment for the disease. The extract which is available for parenteral use may be given intravenously, hypodermically or intramuscularly. There are certain advantages in its use intramuscularly as opposed to the other routes.

In the first place, it is a safer method than the intravenous, because of the fact that one eliminates the possibility of severe reactions, which may occasionally occur following the intravenous use of almost any sort of medication.

There is an advantage in the use of a concentrated solution of high potency for intramuscular administration, as opposed to continuous subcutaneous injection, because of the fact that less frequent injections are necessary, and there is less possibility of infection than there is if the patient himself uses the material.

Because of the several advantages of a concentrated potent solution for intramuscular use, it seemed to me that it might be desirable to attempt to show this by means of a motion picture film. Consequently, instead of attempting to outline these various points in detail, I am going to show a film, which I hope will illustrate these several points. In the first place, one advantage of this material is that the patient is constantly under the physician's direction. That is, for an injection, he must come to the physician, at which time his condition can be ascertained by whatever means is necessary, and follow-up treatment advised accordingly. Then, too, the infrequency of injection makes the treatment an excellent one, from the patient's point of view because he does not have the bother and possibly the difficulty of taking his daily dose of whatever material he may have chosen. There is also a distinct advantage in that this method of treatment produces the most rapid results possible by any method developed up to the present time, and also requires a smaller amount of material, or less frequent injections, than is possible by any of the other methods. Perhaps of greatest importance from the patient's point of view, is the inexpensiveness of this intramuscular form of treatment. That point will be illustrated adequately in the film, and I will not attempt to discourse on that further.

This is the first showing of this film, and I want to apologize for the fact that it is not entirely complete. We have worked ardently on it for several months, and I can assure you that it is no easy matter to make a motion picture film. I have learned many things since we started on this one, although we have had, fortunately, a camera man of experience, who has guided and helped us over some of the pitfalls. There are a few titles lacking in the film, which will be supplied later, but were unfortunately not available for to-day's showing. Notably, in the first reel, I have introduced three pen illustrations to represent, in schematic form, the physiology of blood production. The first scene shows the possible normal mechanism of blood production. The second shows what one might expect in a patient with pernicious anemia, but without the use of liver. The third illustration is the same pernicious anemia individual, but with the introduction of the use of liver in some form, shown in the drawing, as liver by mouth, and illustrating the production of the red blood cells, whereas they were not produced in the second instance.

There are one or two other titles missing, but I think not of sufficient importance to warrant comment. I shall be glad to hear criticisms or suggestions for improvement of the film, if you have any, because we still have a short time in which to complete it for certain other uses, and also, if I have not covered points that are of interest to you by this means, I shall try to answer them later.

Unfortunately, the film is divided into three reels. This is because we have not printed them on the sixteen millimeter stock, which will later bring it down to a two-reel production.

(Dr. Murphy then showed the film on Pernicious Anemia.)

DISCUSSION

PRESIDENT GRAVES: We are most deeply indebted to Dr. Murphy for coming up here and showing us this very remarkable piece of work.

Discussion will be continued by Dr. Joseph D. Shields of Concord.

DR. SHIELDS: There is an expression which our colored folks in the South use that goes like this:

*Read at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 16, 1934.

†Murphy, William P. — Associate in Medicine, Peter Bent Brigham Hospital. For record and address of author see "This Week's Issue," page 942.

"The big folks done spoke, so you might as well hitch-up." I think this well applies in this case.

Since 1926 when Dr. Murphy made his epoch-making discovery in pernicious anemia, we have been looking toward him as a final authority on the subject; nor have we been disappointed to-day. It is very difficult to add to a subject when it is handled as Dr. Murphy has presented this one. I am glad that he has stressed the subject of therapy and particularly the intramuscular treatment. More particularly am I interested in the effects of the intramuscular treatment on the neurological syndrome accompanying pernicious anemia.

Authorities seem to differ as to the relative advantages of the peroral and intramuscular treatment, and some give very little hope for improvement for the neurological syndrome under any circumstances.

You have heard what Dr. Murphy has to say about the therapy. The advantages of the injection treatments seem to be definite. I might review here some of the advantages which this treatment has.

1. It is faster. In the treatment by mouth we expect a reticular response within a period of from four to ten days, while in the intramuscular treatment we often see it within thirty-six to forty-eight hours. The patient who is very weak, has a low blood count and appears on the verge of collapse, often a period of hours after the intramuscular injection gets very striking symptomatic improvements.

2. We have always thought that the patients improved a great deal faster if, at the onset of the treatment, they were given complete rest. With intramuscular therapy, it has been my experience that the patient can be handled as an ambulatory case, with of course some exception in the extreme cases. This is a great help in office practice.

3. As we all know, when we prescribe medicine, oftentimes the patient does not get the complete dosage; but when it is given by needle we are absolutely sure what he gets and therefore can regulate the therapy much more intelligently and positively.

4. In many of our patients we have to consider the economic side of the problem also. The liver extract by needle is much more powerful and considerably cheaper. Three cubic centimeters or the equivalent of 100 grams of liver given by needle will do the work of 5,000 grams of whole liver given by mouth. We know that oftentimes it is difficult for the patient to get liver, particularly those who are not well fixed financially.

5. One of the great disadvantages given for intramuscular therapy is that the patient dislikes to be punctured by needles. However, most patients after the first one or two injections prefer the intramuscular injections once a week to having to take the disagreeable liver extract by mouth. Sometimes we do get rather marked reactions between local and constitutional forms of the first injection. But I think it is important to remember that we are giving a foreign protein and we should handle the case as such. If reaction does occur by the ordinary process of small dose desensitization, we can eliminate these disagreeable reactions.

6. Fewer doses are required with the intramuscular treatment. Oftentimes after having produced our response and obtained a normal blood count, it is necessary to give doses only every three weeks, or occasionally every six weeks.

7. One of the most striking advantages is that some patients who are resistant to liver therapy by mouth, seem to respond to the therapy given by needle. I remember one case particularly that I treated while an intern, who failed to respond to massive doses of liver by mouth. She also failed to respond to ordinary doses by needle. We were considering this case as a possible aplastic type of anemia. When my chief, Dr. Frizzell of St. Luke's Hospital in New York, happened to be talking with Dr. Minot the latter mentioned the fact that a great many of these cases who do not respond to ordinary doses would respond to massive doses. We tried this with very gratifying results.

8. As to the much discussed effect of intramuscular therapy on spinal cord lesions, I feel that we can in a great majority of cases expect some results. I distinctly remember some specific cases when I was working in a blood disease clinic who were resistant to liver therapy by mouth so far as their neurological lesions were concerned, but who responded well to injection therapy. The argument ordinarily put forward against this is that when nervous tissues are destroyed, particularly in the spinal cord, regeneration is impossible and therefore we should not expect symptomatic improvement. As you know, the neurological lesion is a subacute combined sclerosis. There is a degenerative process first of the myelin sheaths and then of the axis cylinders, with fibrotic proliferative repair. However, some authorities have also described in some cases at necropsy a perivascular round cell infiltration somewhat similar to that seen in cephalitis. They raise the question of whether this is an inflammatory process and also raise the question as to whether the damage is due to the effect on the blood vascular supply, or whether it is a semi-inflammatory affair acting directly upon the nerve cells themselves. If in certain cases there is an inflammatory element produced, perhaps by the lack of the intrinsic factor, why should not there be an improvement following large doses of this factor.

It seems to me that although we seldom get striking beneficial results in neurological signs, we do get results in neurological symptoms particularly in paresthesia and in ataxia.

I would like to stress one point in this discussion particularly, and that is the need for adequate dosage. I think before concluding that pernicious anemia, no matter how severe the case, cannot be aided by liver therapy, before surrendering the case as one whose neurological lesions cannot be aided, we should attempt massive dosage of the extract.

PRESIDENT GRAVES: The discussion will be continued by Dr. Clarence O. Coburn of Manchester.

DR. CLARENCE O. COBURN: I wish to compliment Dr. Murphy on his magnificent description, his graphic portrayal of what we have previously considered a deadly, crippling disease. I want to assure him of the gratitude the medical profession has for him for his work in developing the logical, successful treatment of pernicious anemia.

PRESIDENT GRAVES: The subject is now open for general discussion.

DR. JOHNSTON: I want to ask Dr. Murphy two short questions. What, if any place, has hydrochloric acid, and what is the diagnosis for an aplastic type of pernicious anemia?

DR. WESTCOTT: As a matter of diagnosis, I would like to ask Dr. Murphy, when a patient is complaining of indigestion, how frequently upon investigation he finds that it is a pernicious anemia case.

PRESIDENT GRAVES: Is there any other discussion? If not, I will be glad to have Dr. Murphy close the discussion.

DR. WILLIAM P. MURPHY: I am relieved to find that you got something out of the film. I have been quite worried about the possibilities of it, and Mr. Brewster and I have sat up nights attempting to get the legends and titles sufficiently inclusive to explain the pictures, and yet not to use up so much space that it increased the length of the film. As it is, we have progressed from expecting to make one reel up to almost three, and we are having difficulty in keeping it within the limits of two sixteen-millimeter reels. That accounts for many inadequacies in the titles.

It is very difficult indeed to say in a few words just what you want to say and get it all in. However, I think that most of the points are self-explanatory, with the possible exception of one point, which Dr. Scribner has pointed out, and that is in connection with the reticulocyte formation. I think we can explain that a little better. The object in showing the blood smear at two and four days was to show the reticulocyte formation.

Dr. Shields has stressed a number of points, which it seems to me cannot bear too much emphasizing. They are important points. There are many of them which could not even be touched upon in such a picture; some of them we have tried to cover.

The matter of improvement in the cord change and the pessimistic attitude of some who have written and spoken on that subject is one of considerable importance and interest to me, because, as we see our patients with large amounts of treatment, which of course is the way the patient should be treated, we do see improvement, I should say in one hundred per cent of our cases, but not one hundred per cent of improvement in all cases. Certainly one should expect to get anywhere from ten to one hundred per cent of improvement in this cord group of cases. There are, undoubtedly, some patients who have had marked cord change which has persisted for a long period of time before adequate treatment was introduced, and then there is sufficient nerve damage so that the nerve function will improve little. On the other hand, if the patient is treated early and adequately, improvement is really striking. Dr. Shields' point in regard to giving larger amounts of material than are adequate to maintain the blood at 5,000,000 of course is of real importance. It is true that we often give in our cord change cases much more material than seems to be sufficient to maintain the blood at a normal level. But it is interesting to note, also, that very often when the count is almost 5,000,000, and the blood looks normal under the microscope, it may not be normal; that is, actual measurement of the cell volume may show a variation in size. It may be well in some cases to maintain the blood count well above 5,000,000. We have maintained some over 6,000,000 for long periods because of the cord change. But when the count is adequately high and the blood is absolutely normal, that amount of material should be sufficient to maintain the patient in a normal state.

Now the question was asked in regard to the possible advantages of the intramuscular material as opposed to the peroral method of treatment. There are two advantages, in so far as cord change is concerned, it seems to me. The first one is the speed of improvement of the patient. If you can improve the patient's blood rapidly enough to keep the cord disturbance from progressing during relapse, you have accomplished a great deal. We do improve the patient by many days more rapidly than by the peroral method. Then, the other advantage of this method is that one can give as much material as is necessary. You are not limited to your one injection a week. You can give seven injections a week, or ten if you need them. We have no cases which needed, for maintenance purposes, more than two injections a week. Those are isolated cases. But our average clinic maintenance dose, as I showed you on the screen, is one injection in four weeks. In my private practice, I seem to use a little more treatment, that is, my patients perhaps average between two and three weeks between injections. I think that is possibly the result of several factors; one is that my office patients are often much harder working people, from a mental point of view. They are school teachers, lawyers and judges and people who are more active mentally, and have, perhaps, more worries from one or another point of view. Also, some of them have had more cord change in my particular series.

I was asked in regard to the use of hydrochloric acid. I do not use it except for symptomatic reasons, by which I mean indigestion and possibly diarrhea. I cannot see any advantage for it in improving the cord change.

By aplastic pernicious anemia, I presume is meant the increasingly large group of cases that we are seeing that may be diagnosed as atypical anemia. Those cases have features of aplastic anemia, they look like pernicious anemia, and some of them, apparently, develop into a true leukemia. That whole group is a very uncertain one. There is no treatment that I know of up to the present time that does them much of any good, except temporary improvement after transfusion, and, of course, as I say, the prognosis is very poor indeed.

Dr. Westcott brought up the question of the symptoms referable to the digestive tract. Originally in this film, we introduced a series of diagnostic points, mentioning essentially all the points that seemed of importance in making a diagnosis, one of which was the digestive disturbance. This was omitted, however, because of insufficient space in the film.

PULMONARY TUBERCULOSIS ROENTGENOLOGICALLY CONSIDERED*

BY A. S. MERRILL, M.D.†

THE recognition of pulmonary tuberculosis is not the simple matter that some of our clinical colleagues seem to consider it. With its diversity of aspects every pathological condition of the lungs must be considered in its differential diagnosis.

Broadly speaking, two types must be considered: the infantile, a primary infection, and the adult type probably a reinfection.

The infantile or primary type may occur as a small focus in any part of the parenchyma of the lung and is attended by enlargement

*The fourth in a series of papers presented in the Symposium on Pulmonary Tuberculosis at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 15, 1934.

†Merrill, A. S.—Roentgenologist, Elliott Hospital, Manchester, New Hampshire. For record and address of author see "This Week's Issue," page 942.

of the hilus glands draining that area. The original focus may be very small and invisible until healing and calcification take place. The enlarged hilus glands become caseous and may give the impression that they are the site of the infection. Whenever such small calcified spots are seen in the parenchyma there is always an enlargement and calcification of the glands draining the area. Such evidence of healed childhood tuberculosis may be found in most adult chests and does not signify the presence of adult tuberculous infection. The initial lesion may be too small to be seen on the x-ray negative and the suggestive signs are the enlargement of the tracheobronchial nodes. Then they must be differentiated from other types of glandular enlargement and these are found in so many other conditions that often differentiation is impossible. The Committee on Medical Research of the National Tuberculosis Association after long study concluded that the size of the hilum in normal children was so variable that no normal standard could be established. So the majority of childhood infections go unrecognized and, as indicated by the evidence of old childhood infections in adult chests, end in recovery. In children under two years of age from lack of resistance or repeated exposure to infection, the disease often becomes miliary or constitutional with fatal result.

Based on the Roentgen appearances McPhedran has made three classes: 1, parenchymal; 2, tracheobronchial and 3, adolescent.

Parenchymal lesions may exist as a single nodule, massive consolidation of acute caseous pneumonia or miliary tuberculosis. The tracheobronchial type shows by enlarged glands which may or not be calcified. Calcification means an old lesion and may be associated with a parenchymal lesion. The adolescent type approaches the adult type in distribution. The presence of childhood infection may not be discovered until after healing has taken place when it may be seen as small calcified areas. The nature of caseous glands may be revealed only when healing and calcification have taken place. Wherever calcified glands are found, a calcified focus (the Ghon tubercle) is always found in the area drained by those glands. The adult type of reinfection is rarely found in children and when seen it assumes the appearance of adult infection.

In the adult, tuberculous reinfection starts in the apex or the infraclavicular region and either progresses by bronchial extension or heals with fibrosis. Apical lesions increase in frequency with age and are seldom found before ten. If an individual reaches adult life without childhood infection and becomes primarily infected, the infection assumes the character of the infantile type. Such cases do not show the calcification of childhood infection and lack the relative immunity conferred by such early infection. Therefore apical lesions in the adult, which heal by fibrosis, must represent reinfections from an early childhood focus whose primary lesion may have disappeared or remain only as a small calcified spot.

Near the pleural surface the lymphatic drainage is both toward the periphery and the hilum and we should examine carefully this region where engorgement of both lymphatic systems may be found. The lymphatics draining toward the hilum form fan- or cone-shaped areas. Those draining toward the pleura show engorgement by thickening of the pleura indicated by a line of density only visible in a profile view.

The x-ray diagnosis of pulmonary tuberculosis is an anatomical diagnosis of lung involvement. Pleural thickening alone may be called only pleural thickening without knowing the cause. If accompanied by parenchymatous changes, both may be assumed to be part of the same pathological condition and be considered tuberculous. For a definite diagnosis parenchymatous changes are necessary. No single sign in the negative is pathognomonic alone.

DIFFERENTIAL DIAGNOSIS

Pulmonary tuberculosis is so protean in its manifestations that each type should be considered separately.

Childhood tuberculosis shows mainly by glandular enlargement and has nothing characteristic except that it probably will not have the lobulated appearance or size of Hodgkin's disease or lymphosarcoma. All other signs must be correlated. Simple hilum enlargement and increase of peribronchial markings are not enough for diagnosis, as practically any other acute infection will produce the same signs. Children convalescing from some acute pulmonary infection are often sent in for x-ray determination of signs of coexisting tuberculosis, but the diagnosis cannot be justified unless actual parenchymatous infiltration and consolidation are present and then sufficient time should be allowed for the signs of the acute condition to subside and another examination be made to determine the persistence of signs which may indicate tuberculosis.

In the adult, for the diagnosis of incipient tuberculosis, parenchymatous changes must be present. Increased peribronchial markings alone are not enough. Differentiation must be made of acute bronchitis, influenza, asthma, dust inhalation and emphysema. Peribronchial markings extending into the apices are pathological but not necessarily tuberculous. If they are a part of a general peribronchial increase, one must not assume them to be tuberculous. If the increase is confined to the apices it may be tuberculosis but must be accompanied by actual infiltration into the lung tissue. The coexistence of thickened apical or interlobar pleura

is additional evidence. If influenza and dust inhalation can be ruled out, the probability of tuberculosis is strong. Sometimes a single examination is not sufficient in the incipient stage and monthly examinations for several months must be made to note any change in the character of the lesion.

Moderately advanced tuberculosis means involvement of one or more lobes without cavity. It is shown by rounded infiltrations of various size coalescing to form consolidations. It must be differentiated from resolving pneumonia, bronchopneumonia, septic pneumonia, malignancy, dust inhalation, actinomycosis and other fungus diseases.

Fortunately few conditions so frequently involve the upper lobes alone and when no other part of the lung is involved the evidence is characteristic. The resolution of an upper lobe pneumonia may be confusing if seen at a single examination at just the right stage of resolution. Another examination in a few days will show the true nature of the process. Primary tuberculous infiltration in a lower lobe is rare and may not be differentiated from bronchopneumonia or malignancy. The diagnosis of primary lower lobe tuberculosis is not positive unless the bacillus is found. Bronchopneumonia most frequently involves the lower lobes while the upper lobes are the favorite locations for tuberculosis. Septic streptococcus pneumonia will be accompanied by suggestive clinical symptoms which must be known when the negative is interpreted. Secondary malignancy rarely involves the upper lobes and the lesions are coarser and more sharply outlined. They tend to group about the hilum. A primary tumor may be found somewhere. Silicosis may be confusing but has suggestive characteristics and favors the lower lobes. It must not be forgotten that the coexistence of silicosis and tuberculosis is common. Actinomycosis may start at the periphery like tuberculosis and the picture be indistinguishable. It is often chronic, changing little for a long time. The sputum is the only positive differentiation.

Far-advanced tuberculosis is more extensive with or without cavity. It must be differentiated from any condition causing destruction as abscess, bronchiectasis, malignancy, actinomycosis and from localized pneumothorax. Abscess is usually surrounded by a zone of inflammatory consolidation and a fluid level may often be seen. The tuberculous cavity is oftenest seen in an upper lobe while abscess is commoner in the lower areas. Rarely a chronic abscess, especially one caused by Friedländer's bacillus, may be so like a tuberculous cavity that the finding of the tuberculous bacillus is the only absolute proof. Large bronchiectatic cavities are most frequently found near the hilum, and suggestive signs of tuberculosis are not found in other portions of the lung. Lipiodol injection will show the character of the cavities and their relation to the bronchi. Malignant destruction forms irregular cavities surrounded by areas of tumor density and are small compared with the area of the tumor. Actinomycosis and other fungus diseases may form cavities resembling those of tuberculosis or abscess and may be diagnosed only by bacteriological findings.

Acute caseous tuberculous pneumonia is an uncommon type with massive consolidation of one or more lobes and must be distinguished from lobar pneumonia, abscess, primary tumor, massive collapse, bronchostenosis and local effusion. It is most often mistaken for lobar pneumonia. Tuberculous bacilli are not found until destruction begins and differentiation usually depends upon the clinical course and the changes noted by repeated examinations. Primary tumors may show large areas of consolidation with ill-defined borders and marked absence of clinical reaction. Massive collapse will show consolidation, but the displacement of the trachea and heart toward the affected side will give a clue to the condition. Localized pleural effusion may be distinguished only by following the course by repeated examinations.

Fibroid phthisis is a chronic condition with scar formation causing displacement of the mediastinal structures to the affected side and must be distinguished from chronic interstitial pneumonia and massive collapse. Chronic interstitial pneumonia is usually unilateral while in phthisis the other lung usually shows evidence of disease. Massive collapse, the only other condition in which the heart and trachea are drawn to the affected side, is suggested by its acute character and lack of suggestive peri bronchial signs.

Miliary tuberculosis of the lungs will show a general distribution of small soft infiltrations of uniform size and must be differentiated from pneumoconiosis, miliary carcinomatosis, septic pneumonia and bronchiolitis. In all of these the clinical findings, course and history play an important part.

In conclusion I would add that in many cases of lung study the fluoroscopic study of the chest is an important factor in the examination and should be made at the discretion of the roentgenologist when he believes it will add to the diagnostic picture. Also, without going into technical details, the x-ray study of the chest is a complex matter requiring as much knowledge as possible of the different manifestations of lung pathology as well as normal anatomy and physiology, and experience in the interpretation of the characteristics of normal and pathological chest negatives, and especially when interpreting a negative made in another laboratory, as much information as possible should be acquired concerning the past history and clin-

ical findings to correlate with the x-ray findings.

To be of the greatest value to the diagnostician the x-ray negatives should be of the best quality made with the best technical skill by the up-to-date rapid exposure method using 1/20 of a second or less to eliminate completely all motion, even the physiological motion caused by the pulmonary circulation or muscular tremor.

DISCUSSION*

PRESIDENT GRAVES: I am sure that we are all greatly indebted to every one of these gentlemen for the splendid symposium they have given. The subject is now open for discussion, if any one cares to participate.

DR. CURRAN: The New Hampshire Medical Society is indebted to Dr. Overholt and the other experts for the splendid symposium. I wish also to express my personal appreciation, on behalf of the Tuberculosis Committee in our State, for the work he has done with its cases. It may not be known to you, but Dr. Overholt has done this work without fee or compensation, entirely in the interests of the New Hampshire Tuberculosis Committee, by arrangements with the Lahey Clinic. The results obtained, I think, well justify his effort, and I merely wish to express my personal appreciation.

DR. DEMING: *Mr. President*—I would like to express my appreciation publicly to Dr. Overholt and Dr. Lahey for their coöperation in allowing us to give to patients in New Hampshire the benefit of the latest forms of treatment. I think that we should all appreciate it, as medical men, because the members of the medical fraternity are the ones who send the patients to us primarily. I think this whole subject of tuberculosis is very important and too many mistakes are made. That is just criticism and rather frank. I think that the patient should be studied a little more carefully and given a little more of the benefit of scientific opinion by the consulting physician, who, in this case, is his own private doctor.

*This discussion followed the Symposium on Pulmonary Tuberculosis presented at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 15, 1934. The symposium included Dr. Merrill's paper, together with those of Dr. Robert M. Deming (New Eng. J. Med., 211:500, 1934), Dr. S. A. Petroff (New Eng. J. Med., 211:677, 1934) and Dr. Richard H. Overholt (New Eng. J. Med., 211:682, 1934).

MISCELLANY

DEATHS

TRACY — EDWARD A. TRACY, M.D., died at the Elliot Community Hospital, Keene, N. H., following an operation, on September 2, 1934. Dr. Tracy was born at Island Pond, Vermont, on September 19, 1876.

He was graduated from St. Johnsbury Academy in 1895, from Yale College in 1899 and from the McGill Medical School in 1902.

During the World War he was stationed at Camp Dix with the rank of Captain.

Dr. Tracy is survived by his widow, a son and a daughter.

He became a member of the New Hampshire Medical Society in 1907.

MORAN — BERNARD G. MORAN, M.D., for many years a leading physician and a surgeon of Nashua, died at St. Joseph's Hospital in that city on September 26, 1934. Dr. Moran was born in Nashua on March 8, 1870. He was graduated from Holy Cross, attended the University of Vermont, and, in 1891, received his medical degree from the New York University.

Dr. Moran became a member of the New Hampshire Medical Society in 1893. He was a member of the American Medical Association and one of the Board of Governors of the American College of Surgeons. He is survived by his widow and seven children.

Dr. Moran performed the first operation for appendicitis that was done in Nashua.

CROSBY — WALTER T. CROSBY, M.D., of Manchester, died at his home in that city on September 21, 1934. He was born in Laconia in 1871 but had resided in Manchester for the past thirty-two years.

Dr. Crosby was a member of the staff at the Sacred Heart Hospital and was identified with many medical and fraternal organizations. He had been a member of the New Hampshire Medical Society, the New Hampshire Surgical Society, the American Medical Association and the American College of Surgeons. His only immediate survivor is his widow, Mrs. Mollie L. Crosby.

COOS COUNTY MEDICAL SOCIETY

The members of the Coos County Medical Society held a midsummer meeting at the Balsams, Dixville Notch, on September 8. Invitations were sent to the members of the Grafton County Medical Society, the Northeastern Medical Society of Vermont, the Oxford County Medical Society of Maine and the Canadian Societies of Coaticook, Cookshire and Sherbrooke.

Dr. Frederic P. Lord, of Hanover, President of the New Hampshire Medical Society, attended as the official representative of the State organization.

NEW HAMPSHIRE SURGICAL CLUB

The New Hampshire Surgical Club held its thirty-seventh annual meeting at Hanover, N. H., on September 15, 1934. Operative clinics were provided at the Mary Hitchcock Memorial Hospital by members of the staff. In the afternoon, papers were read by Dr. W. J. Paul Dye of Wolfeboro, N. H., and by Drs. David Cheever and Donald Munro of Boston. At the annual banquet in the evening, held at the Hanover Inn, Dr. Elmer H. Carleton of Hanover presided. The speakers at the banquet were Dr. Ernest Martin Hopkins, President of Dartmouth College, and Dr. David Cheever of Boston.

RESIGNATION

ARTHUR C. LISTON, M.D., formerly of North Walpole, N. H., has moved to Bellows Falls, Vermont, and is now a member of the Vermont State Medical Association.

NEW ENGLAND SURGICAL SOCIETY

THE TWO-STAGE OPERATION FOR BLADDER TUMORS*

BY ARTHUR H. CROSBIE, M.D.†

YOU are all familiar and have been for years with the importance of the two-stage operation as a life-saving device in prostatic obstruction. No! I am not forgetting that there is such a thing as transurethral prostatectomy. My statement still stands. In the last few years several of us have brought forward the importance of the two-stage operation for badly infected kidneys where the patient was too septic to warrant a nephrectomy. I think most urologists have accepted this operation as the safest measure in many cases.

Not enough has been said though about the importance of the two-stage operation for bleeding bladder tumors and yet I know that a few conservative urologists do use it occasionally. As it happens I have used it a good many times, perhaps at times needlessly, but on the other hand I am sure that I have saved some lives by its use.

Let me cite a typical case in which I feel the operation should have been used. This happened a good while ago, but it illustrates well what I mean. A man of seventy-one was brought into a hospital with a history of intermittent hematuria of several months' standing. At the time of entering the hospital his bladder was filled with blood clot and distended up to the umbilicus. A Bigelow evacuator was introduced and the bladder emptied and a catheter tied in. After much irrigation the bladder was kept free of blood. After two weeks of drainage the patient's condition had sufficiently improved so that he was cystoscoped. A papillary tumor the size of a hen's egg was seen just above the right ureter. This was fulgurated through the cystoscope and after a few days of uneventful convalescence the patient was allowed to go home to return later for observation.

I asked at the time why an open operation was not done and the tumor entirely destroyed by coagulation with the D'Arsonval current and was told that he had myocarditis and could not stand so serious an operation.

This coagulation entirely stopped the bleeding and after a few days the patient left the hospital.

About two months later this same man was brought to the hospital at eleven o'clock at night with acute retention and the bladder over distended with urine and blood clots. He had been passing clots with difficulty for two days and had finally shut down completely. The Bigelow evacuator was passed and a large amount of blood clot removed and a catheter inserted. In spite of repeated irrigations the catheter did not drain and soon the evacuator was inserted again and clots removed. This was repeated once during the night, but by morning the bladder was again filled with clot. At eight o'clock the next morning in spite of the fact that the patient was in an exhausted condition he was given spinal anesthesia and the bladder opened and the clots evacuated. The growth appeared larger than it did at the time of fulguration two months before.

This tumor was thoroughly coagulated, the whole thickness of the bladder wall being destroyed.

Now let us stop and consider the handling of this case. In developing acute retention associated with bleeding there is a good deal of shock and a good deal of anxiety. There is a certain depressing effect on the kidneys.

It seems to me that altogether too much was done to this man. It is always my principle to relieve the existing acute condition in the simplest possible way. It is well known to all of us that the simplest way to stop intravesical hemorrhage is to empty the bladder and put it at rest. Bleeding from any part of the bladder tends to stop as soon as the bladder is opened, emptied and allowed to collapse. If the bleeding does not stop at once, a small wisp of gauze packed at the bleeding point will stop it.

In the case of the man just cited it might be justifiable to evacuate the clots once and try a catheter if he was in good shape, but I feel that when he filled up a second time the bladder should have been opened at once. It would have been so simple to have opened the bladder under novocaine and evacuate the clots and insert a large tube and have sent the patient back to bed assured that the bleeding had stopped and adequate drainage had been established. With the bladder at rest and draining properly you can wait, the same as you do with the prostate, until the patient is in good shape before doing the radical operation. The wait allows the kidney function to improve. The wait always aids the morale of the patient.

I feel very sure that this two-stage method will frequently be a life-saver. Of course, it means a longer stay in the hospital, but we who have handled prostatic obstruction for many years have learned to disregard long hospitalization when it comes to saving life.

As I mentioned before the bleeding usually stops as soon as the bladder is emptied and

*Read by title before the New England Surgical Society meeting, Burlington, Vermont, September 28, 1934. The author was detained by illness.

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allowed to collapse. Several times I have packed a wisp of gauze against the bleeding point of the tumor, but I think that the bleeding would probably have stopped without it. However, it is always a comfort to know that the bleeding is controlled and there is no harm in putting in the gauze which may be removed a day or two later.

Let me cite a typical case that has come under my observation. One night in February, 1930 a woman of sixty years was brought to the hospital with a history of having passed a good deal of blood in the urine for a month. The afternoon of the day she came to the hospital she had passed clots with difficulty and then had shut down completely. She was seen in the hospital by my associate, Dr. Harold A. Chamberlin, who found her in rather poor condition from loss of blood, and worry. He showed very good judgment by trying nothing palliative, but at once opened the bladder and evacuated the blood clots. He found the bleeding coming from a large tumor. The bleeding was controlled by a light pack and a large tube inserted.

With the bleeding stopped and under adequate drainage the patient improved rapidly and later the tumor was thoroughly destroyed by coagulation, and removed. I am sure that she stood the operation much better after the period of drainage than she would had she had the radical operation at the time she entered the hospital.

I have no doubt that a great many urologists have used the two-stage operation in dealing with bleeding bladder tumors, but it has not been emphasized and I feel that it should be. It is such a simple thing to open the bladder and establish drainage and wait until the patient is in the best possible shape for the more radical operation. Remember that with the bladder open and collapsed, intravesical hemorrhage tends to cease.

THE PRESENCE IN EGG-WHITE AND IN A RICE-POLISHINGS CONCENTRATE LOW IN VITAMIN B_2(G) OF AN ANTI-PERNICIOUS ANEMIA PRINCIPLE

BY D. K. MILLER, M.D.,* AND C. P. RHOADS, M.D.*

CASTLE[1] and his co-workers have shown that in pernicious anemia two factors are required for normal hematopoiesis: first, a heat-labile ferment present in the normal gastric secretion; and secondly, a heat-stable constituent of certain food stuffs, notably meat and yeast. The identity of the second, or dietary factor, has not been established beyond question. The fact that all of the substances which have been shown to contain that factor are also sources of the heat-stable, rat-growth-promoting fraction of the water-soluble vitamin B (vitamin B_2G) led Strauss and Castle[2] to suggest that the two are similar in nature, if not identical.

Wills[3] has recently published an experimental study bearing upon the question, from which it was concluded that the hematopoietic food constituent is not identical with vitamin B_2(G). The conclusion was based upon the fact that a remission could not be effected in a case of pernicious anemia by the administration of an incubated mixture of egg-white extract and normal human gastric juice. It was stated that the egg-white extract used was rich in vitamin B_2(G) and was essentially free from vitamin B_1, but no tests, of the material used, for its content of vitamin B_2(G) are included in the published experimental protocols. Reference to the publication of Chick[4], according to whose method the extract was prepared, reveals the fact that the vitamin B_2(G) content of the extract was probably not very great, since an amount prepared from 25 grams of egg-white was required to supply one rat-growth unit. Moreover, in the experiment published showing the failure of pernicious anemia to respond to the oral administration of the egg-white-extract-normal-gastric-juice digest a possible source of error exists. It was not proved that the patient tested would respond to any orally administered therapy since the period of egg-white-digest feeding was not followed by a similar period during which material of known anti-anemic potency was fed. It was assumed that because an improvement of blood values followed the injection of liver extract a similar improvement would have followed the use of the same material by mouth, a conclusion not in keeping with the findings of Castle[5].

Since the evidence available seemed inconclusive, experiments similar to those discussed have been performed, employing fresh coagulated egg-white as a source of dietary factor. The material used was also tested on rats under standard conditions for its content of growth-promoting vitamin B_2(G). The entire amount of fresh egg-white was coagulated by heating to 100° C. on a water bath. It was then ground through a meat chopper with fine perforations and stored in the ice-box at 4° C. Human gastric juice was obtained by withdrawing all of the fasting gastric contents resulting from the intramuscular injection of 0.5 cc. of a 1:1000

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solution of histamine. A mixture of 100 grams of egg-white and 150 cc. of gastric juice was brought to pH_3 with concentrated HCl and incubated at 37° C. for one hour. The pH was then adjusted to 5.5 with concentrated NaOH and the resultant material immediately fed to patients with pernicious anemia. Prior to the tests of the egg-white-gastric-juice digests, similarly prepared mixtures of known sources of vitamin B_1 and gastric juice were fed as controls of the anti-anemic activity of the gastric juice alone and also after incubation with the anti-neuritic vitamin as a possible source of dietary anti-anemic factor. The preparation of vitamin B_1 employed in Experiment I was the international standard activated Java clay, 15 mg. of which contained one rat unit of the vitamin. In Experiment II the vitamin B_1

TABLE I

PATIENT No. 1. E. R.

Day	R. B. C.	Hgb.	Therapy	Reticulocyte Per Cent
1	1.67	42	25 Gm. Vitamin B_1 incubated with gastric juice	1.0
2			" " " " " " " "	1.0
3			" " " " " " " "	1.2
4	1.49	41	" " " " " " " "	1.8
5			" " " " " " " "	1.6
6			" " " " " " " "	1.8
7			" " " " " " " "	1.2
8	1.49	46	" " " " " " " "	1.4
9			" " " " " " " "	2.0
10			" " " " " " " "	1.4
1			100 Gm. Egg-albumin incubated with gastric juice	1.8
2	1.52	47	" " " " " " " "	2.0
3			" " " " " " " "	1.8
4	1.47	45	" " " " " " " "	2.6
5			" " " " " " " "	4.4
6			" " " " " " " "	5.4
7			" " " " " " " "	*11.8*
8			" " " " " " " "	*9.2*
9	1.60	49	" " " " " " " "	*8.6*
10			" " " " " " " "	4.4
11	2.01	59		3.8
12				3.2
13				2.6
14				1.8
15	2.41	64		1.8

TABLE II

PATIENT No. 2. T. D.

Day	R. B. C.	Hgb.	Therapy	Reticulocyte Per Cent
1	1.51	50	15 Gm. Rice-Polishings Concentrate incubated with gastric juice	2.0
2			" " " " " " " " " "	1.6
3			" " " " " " " " " "	1.8
4			" " " " " " " " " "	1.6
5	1.58	44	" " " " " " " " " "	1.6
6			" " " " " " " " " "	2.8
7			" " " " " " " " " "	3.8
8	1.43	43	" " " " " " " " " "	4.2
9			" " " " " " " " " "	5.4
10			" " " " " " " " " "	6.2
11			" " " " " " " " " "	5.6
12	1.45	42	" " " " " " " " " "	5.4
1	1.31	40	100 Gm. Egg-Albumin incubated with gastric juice	2.4
2			" " " " " " " "	2.8
3	1.49	46	" " " " " " " "	2.6
4			" " " " " " " "	2.2
5			" " " " " " " "	2.6
6	1.41	45	" " " " " " " "	2.8
7			" " " " " " " "	5.4
8			" " " " " " " "	*9.8*
9	1.61	52	" " " " " " " "	7.8
10				*5.6*
11				3.2
12				2.8
13	1.70	55		

source was an acetone extract of rice polishings*, of which 30 mg. contained one rat-growth unit. Two grams of the preparation was the smallest amount which contained one rat-growth unit of vitamin B_2(G). Hence in Experiments II and III the 15 grams of rice-polishings extract administered daily contained 7.5 rat-growth units of vitamin B_2(G) and 1500 units of vitamin B_1. As shown in tables I and II there were no striking increases of the percentages of reticulocytes or improvement of blood values during the control periods when vitamin-B_1-gastric-juice digests were administered. On the other hand, sharp increases of the percentages of reticulocytes and definite improvement of the blood values were effected when coagulated egg-albumin was substituted for the vitamin B_1 concentrate. As soon as a definite remission was established treatment with liver extract was instituted.

Analysis of the reticulocyte percentages obtained in Experiment II presented in table II reveals a mild though quite definite increase during the period of rice-polishings-concentrate-gastric-juice feeding. The maximum obtained was 6.2 per cent on the tenth day, a figure not high enough to be clear-cut evidence of the presence of the dietary anti-anemic factor. The gradual increase in numbers of reticulocytes and the grouping of the higher figures in the period from the seventh to the twelfth days, however, warranted a restudy of this material on another patient.

Experiment III. The same rice-polishings-concentrate-gastric-juice digest was fed to a previously untreated case of pernicious anemia at a low hematological level in a first attack. As is shown in table III a typical and prompt increase in the percentage of reticulocytes took place followed by a sharp rise in the blood levels.

TABLE III

PATIENT No. 3. W. G.

Day	R. B. C.	Hgb.	Therapy	Reticulocyte Per Cent
1	1.54	47	15 Gm. Rice-Polishings Concentrate incubated with gastric juice	1.4
2				1.8
3				2.6
4				2.8
5	1.79	55		8.8
6				12.2
7				26.0
8	2.24	60		25.0
9				12.2
10				4.8
11				4.2
12	2.50	64		3.0

*Furnished through the courtesy of the Burroughs Wellcome Company, Tuckahoe, New York.

The response in this instance was so unequivocal that it seems to be clear evidence that the rice-polishings concentrate employed was a source of dietary anti-anemic factor sufficient to cause a hematological response in a case very susceptible to therapy. It may be assumed that the treatment and incomplete rise obtained in Experiment II were due to the more refractory nature of the disease in that particular individual.

The technic employed for estimating the content of the egg-albumin fed in the rat-growth-promoting vitamin B_2(G) was exactly that described previously by the authors[6]. The basal diet, vitamin-B-free, but otherwise complete, was supplemented by international-standard activated Java clay as a source of vitamin B_1. Coagulated egg-white from the same batch of material which had been tested on the patients was employed as a source of vitamin B_2(G). The results of the tests are shown in table IV. The egg-albumin was adequate to support a normal rate of growth when fed daily in 5-gram amounts. If the daily dose was decreased to one gram, little evidence of growth-promoting activity was encountered. It may be concluded

TABLE IV

5 GRAMS EGG-WHITE + 15 MG. ACTIVATED JAVA CLAY PER DAY

Rat No.	Growth Gain in Grams per Week — Weeks 1	2	3	4	5	Total Given	Average Gain per Week
11	8	8	17	13	11	57	11.4
12	8	6	16	10	8	52	10.4
13	8	10	15	5	4	42	8.4
14	12	4	17	9	16	58	11.6
15	6	—1	7	4	4	20	4.0
16	6	8	15	3	10	42	8.4
17	4	0	6	0	10	20	4.0
18	8	16	26	6	14	70	14.0
19	8	4	11	1	13	36	7.2
20	15	10	17	5	13	60	12.0
	8.3	6.5	14.5	5.6	10.3		9.14
	Average gain per week per rat						

1 GRAM EGG-WHITE + 15 MG. ACTIVATED JAVA CLAY PER DAY

41	5	6	2	10	17	40	8.0
42	4	—2	—2	12	17	29	5.8
43	6	2	—2	10	+		
44	3	1	0	+			
45	8	2	1	+			
46	9	4	4	7	12	36	7.2
47	10	3	0	0	+		
48	4	0	2	+			
49	6	2	4	10	6	28	5.6
50	8	0	4	14	8	34	6.8
	6.3	1.8	1.3	10.5	12.10		6.68
	Average gain per week per rat						

from these tests that the particular material employed was a source, though not a particularly good one, of the heat-stable, growth-promoting vitamin $B_2(G)$.

The observations presented indicate that, contrary to the conclusion of Wills, the dietary anti-pernicious anemia factor is present in egg-white. Hence, they fail to substantiate Wills' conclusion that the anti-pernicious anemia factor and vitamin $B_2(G)$ are dissimilar. Clearly, the positive conclusion, namely, that vitamin $B_2(G)$ and the anti-pernicious anemia dietary constituent are identical is also not justified, since egg-white may well contain an almost infinite number of substances other than the vitamin. Furthermore, the results of Experiment III, in which a clear-cut increase of reticulocytes and improvement of blood values followed the administration of an amount of rice-polishings concentrate containing only 7.5 units of vitamin $B_2(G)$ are clear-cut. They again suggest that adequate evidence of the identity of that material with the anti-anemia factor is not at hand. If vitamin $B_2(G)$ is the active principle it must be effective in an exceedingly low concentration since in Experiments I and II no more than 100 and possibly as few as 20 units daily were sufficient to effect a response. In Experiment III an amount of material containing only 7.5 units daily was effective. The difference in weight between the largest rats employed and an average adult human being is as 1:1400. Conclusive proof of the identity or lack of identity of the dietary anti-anemia factor and vitamin $B_2(G)$ clearly must be deferred until isolation of the vitamin in a pure form has been effected.

CONCLUSIONS

1. Egg-white, containing one rat-growth unit of vitamin $B_2(G)$ in 5 grams is a source of dietary anti-pernicious-anemia factor when 100 grams are fed daily after incubation with 150 cc. of normal human gastric juice.

2. Rice-polishings concentrate containing one rat-growth unit of vitamin $B_2(G)$ in 2 grams is a source of the same factor when 15 grams are fed daily after incubation with 150 cc. of normal human gastric juice.

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LEVERAGE REDUCTION IN FRACTURES OF THE SURGICAL NECK OF THE HUMERUS

BY FREDERIC JAY COTTON, M.D.,* AND GORDON MAC KAY MORRISON, M.D.*

IT is the fashion to treat fractures of the surgical neck of the humerus in abduction, and in these days of traction, continuous traction is used on these also.

There is a place for traction treatment in abduction and a not very large place for "aviation" splints and other ambulatory abduction apparatus. But just as in many fractures of the anatomical neck of the humerus, it is often wise to refrain from any attempt at reduction and get them moving; so, too, with the surgical neck cases, if they are once reduced, the very early beginning of "active assisted" motion is the one thing needful.

The tendency to displacement, once the fracture is *exactly* reduced, is apt to be almost nothing. Remember that these are predominantly fractures of the elderly and those with weak or atrophied muscles. And if one is going to start motion really *early,* it can be started from a position at the side just as well as from the very uncomfortable position of right-angle abduction. Real reduction is necessary, however, and this we propose to consider. This does not mean reduction with elaborate traction machinery. It means intelligent leverage with hand traction, *after* prolonged traction under anesthesia has abolished muscle spasm, for the time at least.

In nearly all these cases the break is roughly transverse, the displacement that of the upper end of the distal fragment *upward* and *inward.* The strong pectoral muscle maintains the displacement. Therefore, reduction means anesthesia. It also means traction—temporarily, but for reduction only.

The form of reduction we have used, latterly, is as follows: first, examination with the fingers as well as the x-ray film to define the relation of fragments. Then the patient is anesthetized, preferably close to an x-ray machine, and placed on his back. Strong steady pull is exerted downward on the wrist (arm in extension) with a hand grip or with a clove hitch

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about the wrist, for at least five minutes, by the assistant. Then the operator thrusts his arm through the axilla and, with his hand flat on the table beneath the patient's scapula, uses his forearm as a lever to pry the humerus outward. This is to overcome the spastic pull of the great pectoral muscle. When this spasm eases off, the time for reduction has come. The assistant *ad*ducts the straight arm to the midline and forward (upward as the patient lies) while the surgeon, with the position of his arm almost unchanged, uses this arm as a lever to pry the upper end of the lower fragment *out* and *backward*.

After one or two maneuvers of this sort, something moves. Then a check x-ray film is taken, and the maneuver repeated or varied as indicated.

It is astonishing what relatively exact repositions may be secured in this simple way.

As above noted, if reposition is fairly exact, the tendency to redisplacement is but small, and no apparatus other than a sling and a circular swathe is needed.

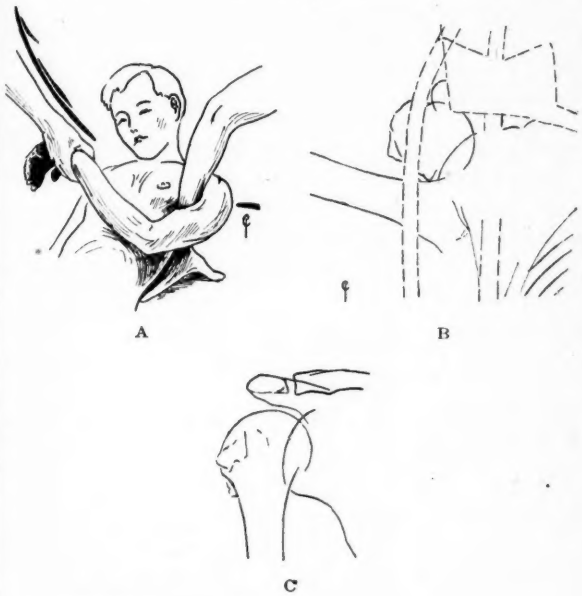
Motion may be begun early, and early means *one week, not four*. Most useful are pendulum exercises with the arm hanging free, the patient doing the swinging.

Three typical cases are cited, as ordinary routine examples.

One, a woman of fifty, was reduced December 20, 1933, and active motion begun January 3, 1934. Mobility was complete at eight weeks.

The second, a man of fifty-seven, was reduced January 9, 1934. There was motion at one week. This was a little slower in limbering up, and required about three months to produce full function and range of motion.

The third, a woman of forty-five, was first seen two weeks after injury. Data are given in the "legend" of the illustration. The results at two months were excellent despite the delay and repeated previous trauma in attempts to reduce.



A. Diagram of reduction.
B, C. Shoulder two weeks after injury March 3, 1934, following two reductions, in abduction splint, redisplaced, presumably, by leverage on the fulcrum of the tight pectoral muscle.
Same shoulder reduced as above described, and put up in the natural position. There was no recurrence of displacement, and early motion secured an excellent result.

MEDICAL PROGRESS

PROGRESS IN TUBERCULOSIS

BY JOHN B. HAWES, 2ND, M.D.,* AND MOSES J. STONE, M.D.*

FROM a perusal of the literature of the past year it is evident that the attention of clinicians is mainly focused on collapse therapy, especially with its clinical application and technical methods. As a result, our conception of when and how collapse therapy should be applied has been greatly clarified and placed on a more scientific basis. While enthusiasts have somewhat subsided in their vigorous attempt to collapse every unilateral lesion, skeptics are now fewer in number. All of which leads to progress.

Laboratory studies have been given more attention than in previous years. The sedimentation rate for instance has been found to be a delicate indicator of the activity of disease preceding other symptoms and signs of activity. In many institutions it is becoming a routine procedure. The differential white count, especially the study of the monocyte and lymphocyte ratio, is also a fairly well-established indicator of activity and progression of the disease. Increase in the sedimentation rate along with an increase in the number of monocytes is now considered to mean active disease and has proved so far to be a more delicate test of this than any other measures that we have at hand even preceding roentgenological findings in the minds of many.

The subject of the childhood type of tuberculosis is gradually being clarified. The theory that this represents the primary infection and is the causative agent in the production of allergy rather than immunity is fast gaining ground. Most writers now agree that adult tuberculosis is due to reinfection in a previously allergic individual.

The subject of vaccination with B.C.G. is for-

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tunately now in the hands of trained investigators so that before long some really scientific data on this will be available. In this country this is still in the experimental stage, its use being largely in the hands of special investigators, mainly Park in New York and Petroff at Saranac Lake.

Much light has been thrown on the subject of pneumonoconiosis. The relationship of the silica dust and tuberculosis is well established. On the other hand, other dusts have been found to bear very little relationship to tuberculosis. With greater knowledge and greater crystallization of our ideas concerning pneumonoconiosis there should eventually be presented some very practical measures which should lead to the prevention of this serious disease.

PATHOLOGY

Dickey, L. B., and Starks, D. J.: (*J. Pediat.* 1:674 [Dec.] 1932) contrary to our former ideas on this subject find that most children can handle moderate amounts of tuberculous infection better than adults, providing continuous reinfection is prevented and the children immediately hospitalized.

Stivelman, B. P.: (*J. A. M. A.* 99:1666 [Nov. 12] 1932) believes that lobular or confluent lobular atelectasis is a most frequent occurrence in all stages of pulmonary tuberculosis. He feels that the narrowing of the interspaces, deviation of the mediastinum toward the lesion and elevation of the diaphragm in early cases are not due to fibrosis, which is of slow evolution, but to lobular atelectasis, which is apt to develop with great rapidity in any tuberculous pulmonary area. He adds that the great frequency of this type of atelectasis in pulmonary tuberculosis indicates that it is one of nature's ways of initiating fibrosis in the involved area.

Sayé, L.: (*Arch. Méd.-Chir. de l'app. Respir.* 7:43, 1932) is of the opinion that massive collapse occurs more commonly as a complication following hemorrhage in early tuberculosis than in advanced tuberculosis. He explains that atelectasis supervenes only as the result of two factors, first, the presence of a bronchial obstruction, and secondly, the release of a reflex spasm set up in a healthy bronchus by the foreign body, a power lost in cases of diseased bronchi of long duration. Thus, the possibility of massive collapse must be kept in mind in caring for patients with recent tuberculous disease whenever hemoptysis occurs.

Fraenkel, A.: (*Beitr. z. Klin. d. Tuberk.* 81:692 [Dec.] 1932) takes up the subject of the early roentgenological diagnosis of pulmonary tuberculosis. For years a slight area of infiltration was considered to be the first clinical and roentgenological sign of pulmonary tuberculosis. He, however, is of the opinion that these early infiltrations are exudative lesions with central caseation, and for this reason can not be the earliest lesions. He feels that a typical manifestation of true incipiency is the roentgenological picture of single, round, well-circumscribed foci, which are at times seen in x-ray films. These are usually in the infraclavicular region, 1 to 4 cm. in diameter, and appear as soft shadows surrounded by apparently normal parenchyma. They may develop into the characteristic picture of infiltration with or without excavation.

Herrnheiser, G.: (*Beitr. z. klin. d. Tuberk.* 81:720 [Dec.] 1932) points out that small initial shadows on the x-ray film correspond exactly with the involvement of one lobule. When a whole lobule is involved, the contours are sharp and relatively straight by virtue of the limiting interlobular septum. He stresses greatly the importance of proper x-ray technique.

Vercesi, R.: (*Lotta contro la tuberc.* 3:1095 [Oct.] 1932) believes that the retrogression of lesions in the contralateral lung in cases of artificial pneumothorax is due to the partial indirect compression of the contralateral lung brought about by displacement of the mediastinum. The resulting diminution of toxins from the compression of the more extensive and active foci, increases the disease resisting powers of the body, so that it is better able to heal the smaller lesions in the non-collapsed lung.

Haywood, E.; Morriss, W. H., and Wilson, G. C.: (*Am. Rev. Tuberc.* 28:637 [Nov.] 1933) studied tuberculosis in families of sanatorium patients. They obtained positive tuberculin tests in 91.8 per cent of all individuals examined, varying from 74.2 per cent in the age group under five to 100 per cent at thirty years. From x-ray examination 8.4 per cent were classified as having manifest tuberculosis, 29.3 per cent as latent, 62.2 per cent as negative. The highest percentages of manifest lesions were found in the age group of fifteen to nineteen, i.e., 14.2 per cent and in the group of individuals over sixty, 16.6 per cent. Comparison between cases in contact with patients with a positive sputum and those negative showed a considerably greater incidence of manifest tuberculosis among the sputum positive group, but no appreciable difference as regards latent or negative cases. Living conditions and nativity did not show any appreciable effect on the incidence of tuberculosis. They conclude that the chief danger to children lies in families in which the source of infection is in either fathers or mothers.

Myers, J. A.: (*Am. Rev. Tuberc.* 28:793 [Dec.] 1933) takes up the rôle of first infection and reinfection types. He states at the beginning that infection is an unwelcome guest in any case leaving behind a trail of undesirable conditions. His contention is that the first infection type of tuberculosis is a benign condition with lesions which vary in size and num-

ber and which tend to heal. Such infections, however, may at any time liberate bacilli into the blood stream or upon allergic tissues which may result in rapidly progressive miliary tuberculosis or in chronic disease of the lungs, bones and joints, or other parts of the body. He feels that any immunity which the first infection type may confer is incomplete in character. He, therefore, makes the assertion that we are no longer justified in speaking of tuberculous infection without disease, claiming that they are one and the same thing. He feels that employment of epidemiological measures holds the same promise for tuberculosis as it does for other communicable diseases.

Evarts, H. W.; Potter, R. J., and Dunn, E. G.: (*Amer. Rev. Tuberc.* 29:123 [Feb.] 1934) in a study of children of tuberculous parents found that the tuberculosis mortality rate in the age group of fifteen to thirty-nine years was two or three times as high as that of individuals not so exposed.

Blum, R.: (*Beitr. z. klin. d. Tuberk.* 82:744 [May 22] 1933) reports aggravation of a preëxisting but unrecognized pulmonary tuberculosis by violent trauma to the chest. In each the causal connection was established by hemoptysis shortly following the trauma. (Such trauma need not be violent, nor need there be a hemorrhage to establish a causal relationship in such cases. J. B. H.-M. J. S.)

Leggett, E. A.; Myers, J. A., and Levine, I.: (*Am. Rev. Tuberc.* 29:348 [March] 1934) believe that spontaneous pneumothorax is more frequent than a study of the literature would lead one to expect. They emphasize that many but not all spontaneous pneumothoraces are tuberculous in origin. They feel that the diagnosis of spontaneous pneumothorax must be confirmed by the aspiration of air from the pleural cavity or by x-ray films or fluoroscopic examination. A higher percentage of idiopathic spontaneous pneumothoraces is seen in office and consultation practice than in hospital or sanatorium practice.

(It is quite possible to make a clinical diagnosis of this condition on physical signs and symptoms alone. J. B. H.-M. J. S.)

Stewart, C. A.: (*Am. J. M. Sc.* 185:346 [March] 1933) is of the opinion that the relationship existing between the childhood and the adult type of tuberculosis consists largely in the tendency for the primary infection to prepare the patient for the development of phthisis should he perchance later experience a reinfection of sufficient severity to produce an intrapulmonary lesion. He believes that phthisis does not usually result from a lighting up of an old infection received in childhood. The part the childhood infection plays in favoring the later development of phthisis depends more upon sensitizing the soil so that later on it reacts to the tubercle bacillus and its products rather than upon a lighting up of the old disease acquired during childhood.

Coryllos, P. N.: (*J. A. M. A.* 100:480 [Feb. 18] 1933) made a study as to why and how rest and collapse therapy cure pulmonary tuberculosis. He points out that tubercle bacilli are not only strict aerobes, but they need a large amount of oxygen for their continuation of life and growth. If, therefore, conditions of anaerobiosis are created in a tuberculous area of the lung or of any other organ, tubercle bacilli cannot continue to thrive and grow. He shows that when a large bronchus is obstructed, the air entrapped in the territory of the lung corresponding to that bronchus will be completely absorbed. Thus, he feels that "kinking" of the bronchial outlets of the cavity, for instance, will bring about its eventual closure, which explains the occasional spontaneous disappearance of cavities and more especially their collapse and cure after successful pneumothorax or thoracoplasty.

Stanbury, W. S.: (*Am. Rev. Tuberc.* 29:528 [May] 1934) describes the anatomical changes in the diaphragm following phrenicectomy. He gives an analysis of eleven cases of atrophy of the diaphragm. He found evidence of atrophy as early as the third week after section of the phrenic nerve and complete atrophy by the fourth month. After paralysis, one-half of the diaphragm is elevated and eventrated into the thorax, which, because of stretching becomes a thin whitish membrane of parchment-like thickness. Histologically he found the atrophy to be complete and uniform. The author warns that in view of the marked distortion of the abdominal viscera in ten of his eleven cases, three of which had a fatal gastroduodenal obstruction, the possibility of complications must be considered when advising phrenicectomy for the treatment of pulmonary disease.

LABORATORY STUDIES

Crimm, P. D.; Strayer, J. W.; Watson, H. S., and Heimann, G. G.: (*Am. Rev. Tuberc.* 28:202 [Aug.] 1933) studied the effect of viosterol on the absorption, retention and excretion of calcium. They found that there is a direct correlation between the amount of the calcium intake and the fecal calcium output. During the early administration of viosterol, however, there is a decrease in urinary and fecal calcium. Continued administration increases the urinary calcium and decreases the fecal calcium. On the whole, they found that viosterol definitely increases the absorption of calcium.

Arnold, L.: (*Am. Rev. Tuberc.* 28:262 [Aug.] 1933) finds that the increase in deaths from tuberculosis in young females over males of the same age group is due to a biological change in the female. The zone of tissue around a tuberculous focus is stimulated by the normal

metabolic changes taking place in the female between ovulation and menstruation. He found that the alteration in the metabolic rate, permeability of capillaries, the increased cellular exchange and tissue digestive processes create an adverse environment around such a focus, which accounts for the increased death rate.

(This is interesting if true. J. B. H.-M. J. S.)

Friedman, E.; Black, M. H., and Esserman, A. L.: (*Am. J. Dis. Child.* 45:58 [Jan. 1] 1933) made a comparative study of tuberculin tests in children. They found the Pirquet and the modified Moro (percutaneous) tests to be unreliable. For routine office and bedside work, the "single puncture" method is to be preferred, in that it is easier to perform, requires less time, does away with the necessity for dilution and refrigeration, and is less expensive.

Duffy, J.: (*Tubercle.* 14:60 [Nov.] 113 [Dec.] 1932) has tried to evaluate various laboratory methods, as an index to activity. The leucocytic reactions and especially the proportion of lymphocytes to monocytes appear to be the first indication of pathological activity. This, however, is too delicate an index for practical purposes. The increase in blood sedimentation he believes to be a coincidence and sequel of spread of the disease, rather than an antecedent or early manifestation of impending trouble. He concludes that in the estimation of activity in pulmonary tuberculosis the order of importance is first the leucocytic reaction, secondly, blood sedimentation, thirdly, x-ray and lastly, clinical signs and symptoms. He warns, however, that this order is not absolute and a correlation of data is imperative.

(We are still of the opinion that a careful study of clinical signs and symptoms is the best criterion of activity. J. B. H.-M. J. S.)

Soper, W. B.; Alpert, L. K., and Adams, M. S.: (*Am. Rev. Tuberc.* 28:667 [Nov.] 1933) make a comparative study of the vaccinating property of heat-killed tubercle bacilli and compared it with that of B.C.G. and living human tubercle bacilli. They found that the rabbits vaccinated with heat-killed bacilli appeared the best protected, as judged by both survival and autopsy findings. The animals vaccinated with living human bacilli and B.C.G. were about equally protected. They feel that the heat-killed bacilli possess greater antigenic properties than do the heat-killed "undissociated" bacilli and offer vaccinating possibilities which merit further study.

Oatway, W. H.: (*Am. Rev. Tuberc.* 29:73 [Jan.] 1934) made an interesting follow-up study three and a half years later on a group of patients who had leucocyte studies in 1929. He finds that previous conclusions as to the meaning and the relative seriousness of the types of leucocyte pictures have been further reaffirmed and that the leucocyte count actually reflects the pathological process of the tuberculous individual.

Siltzbach, L. E.: (*Am. Rev. Tuberc.* 29:673 [June] 1934) in a study of the sedimentation test on 494 patients with pulmonary tuberculosis feels that this reaction indicates the progress of the disease, and that a change in the reaction often precedes a change in clinical symptoms and signs. It is of value, therefore, in prognosis. In collapse therapy this test may be used as a gauge of the effectiveness of treatment. He strongly recommends it in routine sanatorium and private practice work.

CHILDHOOD TYPE TUBERCULOSIS

Myers, J. A.: (*Am. Rev. Tuberc.* 28:93 [July] 1933) reaffirms his beliefs concerning the rôle of reinfection in pulmonary tuberculosis. Students of nursing and medicine at the University of Minnesota go through a routine tuberculin test and a roentgenographic chest examination. On the basis of a very extensive study he found that first infection with tubercle bacilli as determined by repeated tuberculin tests occurring in adult life, resulted in no serious illness. Therefore, the development of rapidly progressive and highly destructive disease in adults who have not been previously infected is apparently a myth. The first infection type of tuberculosis in the adult is a benign disease which comes under control just as well as it does in the infant and the child. His main point is that following the first infection type of tuberculosis, the adult like the infant and child, becomes a potential case of the reinfection and the destructive forms of tuberculosis. He concludes that a positive tuberculin reaction, indicating allergy resulting from first infection type of tuberculosis, is a definite liability, since the destructive forms of tuberculosis do not ordinarily develop in its absence. Therefore, the longer it can be postponed the better.

Johnston, J. A., and Chadwick, H. D.: (*Am. Rev. Tuberc.* 28:244 [Aug.] 1933) have studied the childhood type of tuberculosis in Detroit. They found an incidence of reaction to tuberculin of 14.3 in the age group from birth to fifteen years. The incidence in the age group five to fifteen years was 17.9 per cent. During the first four years of the study two adult type cases have developed out of 359 reactors only, each of whom had been diagnosed previously as having the childhood type of tuberculosis.

Korns, J. H.: (*Am. Rev. Tuberc.* 28:251 [Aug.] 1933) feels that tuberculin testing of large numbers of elementary rural school children is time consuming and expensive, and that the infrequency of tuberculosis in this group of children hardly warrants the routine use of this method. He does, however, urge tuberculin-testing of high school students in the hope that by x-raying the reactors adult lesions may be

discovered before illness causes the student to seek medical advice. He advises that x-raying of the lungs should not be withheld when the tuberculin test is not allowed.

Bridge, E., and Stokes, A. M.: (*Amer. Rev. Tuberc.* 28:522 [Sept.] 1933) compare the results obtained in examining 11,296 children at their schools with those obtained by examining 2458 contact cases of school children at the dispensary where they were brought because of such contact or because of some symptoms suggesting ill health. The incidence of tuberculosis was four times as great among this latter group as among supposedly healthy rural school children. Ten times as many cases of tuberculosis were found in the dispensary group, as in the school-examination group. They demonstrate the superiority of the dispensary examination as a method of case findings.

Hetherington, W. H.; McPhedran, F. M.; Landis, H. R. M., and Opie, E. L.: (*Am. Rev. Tuberc.* 29:142 [Feb.] 1934) give their results of a further study of tuberculosis in public school children. They find a very high percentage of reactors. In the age group fifteen to nineteen years 79.6 per cent of white boys, 87.9 per cent of white girls, 87.9 per cent of colored boys and 86.4 per cent of colored girls reacted to tuberculin. The incidence of serious tuberculous lesions was approximately 0.3 per cent in white children five to eleven years of age and more than five times as frequent in colored children of the same ages. The incidence of tuberculous lesions in children twelve to twenty years of age was 1.0 per cent for white boys and 2.3 per cent for white girls.

Morgan, R.: (*Am. Rev. Tuberc.* 29:577 [May] 1934) emphasizes the fact that moderately advanced and advanced pulmonary tuberculosis in children and adolescents is an extremely serious disease. He finds that the vast majority of these cases will die despite prolonged bed treatment. However, he finds that early or incipient cases do very well under bed treatment. He calls attention to the necessity of educating the general public as to the urgency for prompt treatment in every child in whom a diagnosis of pulmonary tuberculosis has been established.

COLLAPSE THERAPY

Schwatt, H.: (*Am. Rev. Tuberc.* 28:165 [Aug.] 1933) suggests bilateral phrenic exeresis in cases of chronic bilateral disease. He warns that this is a very serious procedure and that it is applicable to a very limited group of prognostically unfavorable cases.

Fischel, K.: (*Am. Rev. Tuberc.* 28:411 [Oct.] 1933) discussing the surgical treatment of cavities points out that the peripheral cavity is an indication for immediate collapse. By the early use of pneumothorax in infiltrations that do not retrogress in a short time, the formation of cavities and of adhesions may be prevented. He stresses the point that the suspected cavity should be recognized and the distention of the cavity by adhesions and pneumothorax should be avoided. He feels that phrenicectomy is applicable for the obliteration of cavities if compression by pneumothorax proves unsuccessful. He finds that it can be used to great advantage in conjunction with incomplete pneumothorax, or in partial or total thoracoplasty.

Ornstein, G. G., and Ulmar, D.: (*Am. Rev Tuberc.* 28:484 [Sept.] 1933) discuss the question of treatment of the acute forms of tuberculosis. They maintain that the exudative forms tend to clear spontaneously, provided there is no reinfection. They emphasize, therefore, that in these cases no interference is needed. The caseous pneumonic form on the other hand can be combated only by the mechanical method of compression therapy. The index of success is the disappearance of tubercle bacilli from the sputum. According to the authors, phrenic nerve operations are useless. They use artificial pneumothorax, thoracoplasty and apicolysis, when compression therapy is indicated.

Leslie, G. L.: (*J. A. M. A.* 100:313 [Feb. 4] 1933) gives the various surgical-collapse measures in the treatment of 420 patients with the adult type of pulmonary tuberculosis, consecutively admitted to the Michigan State Sanatorium. Of these a total of 324 or 77.14 per cent were selected for collapse therapy. He concludes that the majority and not the minority of patients with active pulmonary tuberculosis are suitable for collapse therapy in some form, and that considerable involvement of the better lung even with cavities present, does not necessarily contraindicate such therapy. He also emphasizes that such therapy should be considered from the standpoint of immediate application as soon as possible after a diagnosis is made, and should not depend upon delayed admission to institutions with long waiting lists.

Hawes, J. B. 2nd, and Stone, M. J.: (*J. A. M. A.* 98:2048 [June 11] 1932) present an analysis of the extent to which collapse therapy is used in the treatment of pulmonary tuberculosis in the New England States. They found that only 4.2 per cent were selected for some form of collapse therapy, against 15 to 57 per cent in other parts of the country, especially in health resorts. The authors recommend that (1) tuberculosis and its diagnosis and treatment should be given distinctly more attention in medical schools than is now the case; (2) every general hospital, large or small, should freely and willingly admit tuberculous patients for study, diagnosis and operative treatment if necessary; (3) every tuberculosis hospital and sanatorium should have an x-ray plant or easy access to one and should be otherwise equipped

to carry on compression treatment, while the members of its staff should be trained in the selection of cases and in the technique of pneumothorax; and (4) the subject should form part of the yearly program of every county and local medical society.

Burrell, L. S. T.: (*Lancet*, 1:356 [Feb. 18] 1933) warns that in the treatment of cavities the patient should not be subjected to treatment which is more dangerous than the cavity itself. He feels that the annular shadow often shown by radioscopic examination of tuberculous lungs is in the majority of cases probably due to a cavity. In the absence of any symptoms or signs this type of cavity does not need any special treatment. In the acute cases with cavity formation artificial pneumothorax is the best treatment. Failing this, the lung should be rested by sandbags. Phrenic evulsion and thoracoplasty are contraindicated.

Peters, L. S.: (*Ann. Int. Med.* 6:802 [Dec.] 1932-33) believes that the man who temporizes with tuberculosis and who treats advanced disease by bed rest alone with the usual routine of twenty-five years ago, failing to utilize the methods of collapse therapy at his command, is a menace to his patients and is not giving the best the profession has to offer in the treatment of this disease.

(Such plain talk as this will do much good. J. B. H.-M. J. S.)

O'Brien, E. J.: (*J. Thoracic Surg.* 1:603 [Aug.] 1932) states that the incidence of pneumolysis in his clinics is very small. Phrenicectomy often releases the tension of a taut adhesion so as to permit closure of a cavity. Of the two methods of severing adhesions, the author prefers that of rib resection and incision of the pleura rather than the method of Jacobeaus. The advantages of this operation include increased accessibility to adhesions, and a decreased danger of hemorrhage.

Armand-Delille, P.; Hechter, P., and Magrin, A.: (*Rev. de la Tuberc.* 12:763 [July] 1932) established twenty-four unilateral collapses in infants with good results in eighteen. Fifty-seven unilateral and twenty-seven bilateral collapses were instituted in older children, and fifty-four and seventeen good results respectively were secured. They conclude that collapse therapy is incontestably the most efficacious aid, entailing no significant dangers or risks, in the treatment of pulmonary tuberculosis in childhood.

Haynes, J. N.: (*J. Thorac. Surg.* 11:34 [Oct.] 1932) emphasizes the fact that the use of oleothorax in pulmonary collapse requires a very careful technique. Of 150 collected cases there were thirty-nine deaths, some of which were directly attributable to the technique. He advises the injection of only 5 cc. of oil at first, in order that the reaction of the pleura may be tested. Repeated doses should not be administered until the reaction has subsided. He feels that it is necessary to follow these cases closely, since the injection of oil is often followed by varying degrees of serous effusions.

Patronikola, G. E.: (*Beitr. z. klin. d. Tuberk.* 81:600 [Nov.] 1932) found that phrenicectomy resulted in only 4 per cent cures and in 4 per cent improvement. Since this percentage is not more than could be expected without surgical interference he concludes that it is of doubtful therapeutic value as an independent operation. He states, however, that the operation is probably indicated in a small number of well-selected patients.

Allen, D. S.: (*J. Thorac. Surg.* 1:587 [Aug.] 1932) reports a case in whom he performed bilateral thoracoplasty. The upper five ribs were removed from the left chest, beginning at the transverse process and extending around to the mid-axillary line. Twenty-seven months after the left thoracoplasty, a partial upper right thoracoplasty was done. Satisfactory improvement resulted and ten months later, the patient returned to work. Following the second operation the vital capacity was 2075 cc.

Pollock, W. C.: (*Amer. Rev. Tuberc.* 29:284 [March] 1934) recommends the more frequent use of bilateral compression therapy. Among the various combinations aside from bilateral pneumothorax he mentions, (1) pneumothorax with contralateral phrenic exeresis: (2) thoracoplasty with contralateral pneumothorax; (3) oleothorax and pneumothorax contralaterally. These forms of therapy have greatly increased the case group to which compression treatment may be applied. He feels that the once greatly limited field of collapse therapy has been broadened to a field with practically unlimited possibilities. On the whole, he found that the failures and the severe complications encountered were greatly overshadowed by successful results.

Mayer, E.: (*J. A. M. A.* 100:1478 [May 13] 1933) stresses the fact that complete and early closure of pulmonary cavities excreting tubercle bacilli is imperative, as there is continual danger of hemorrhage, spread by extension, aspiration pneumonia and visceral degeneration. He finds that early cavities of medium size, surrounded by little disease, disappear in 50 per cent or more of patients on strict bed rest and that cavities regardless of their age may heal in 40 per cent of patients under more prolonged bed rest. He finds that 65 per cent of patients with cavities are still living at the end of five years and that old upper-lobe cavities with good drainage, especially in elderly patients are consistent with prolonged life. On the whole, however, he finds that 80 per cent of patients with cavities closed, symptom free and sputum negative, as a result of satisfactory collapse of the lung, regain complete working capacity; those

with cavities uncollapsed and sputum positive rarely make an economic recovery.

McMahon, B. T. and Kerper, E. H.: (*Am. J. M. Sc.* 186: 170 [Aug.] 1933) make a strong plea for individualization in the treatment of pulmonary tuberculosis with cavitation. The presence of cavity undoubtedly constitutes a serious menace, but they urge that before instituting collapse therapy all factors be carefully weighed. If circumstances are not highly unfavorable, a preliminary period of observation with the patient at bed rest is fully justified. Of 296 patients with cavities, 22 per cent secured spontaneous closure of cavity.

Johns, F. S.: (*Am. J. Surg.* 20: 737 [June] 1933) pleads for selective collapse when thoracoplasty is carried out. He finds that a less extensive operation is often of equal value and is less hazardous. He advises the removal of sections of the upper five to seven ribs only. Occasionally he supplements it with phrenic-nerve surgery.

Bluhm, I.: (*Beitr. z. klin. d. Tuberk.* 82: 355 [March] 1933) reports three cases in whom it was possible to establish a pneumothorax again after a previous pneumothorax on the same side had been abandoned. The primary pneumothoraces lasted three years, six months, and seven years respectively. The interval between the first and second pneumothoraces was eight years, nine months, and three and one-half years, nine months, and three and one-half years respectively. Two of the cases showed many adhesions. After endopleural cautery a satisfactory pneumothorax was established in two cases.

Noack, R.: (*Beitr. z. klin. d. Tuberk.* 82: 397 [March] 1933) gives the following indications for phrenicectomy: (1) Recurrent or continuous hemoptysis, if pneumothorax, partial thoracoplasty or tamponing cannot be used. (2) In cases in which there are contraindications against thoracoplasty or tamponing, such as too far advanced age, arteriosclerosis, bilateral disease, cardiac disease. (3) To improve the action of pneumothorax which is incomplete on account of adhesions, and only in such cases in which pneumolysis or thoracoplasty can not be performed. (4) In patients who refuse thoracoplasty but who are willing to try phrenicectomy. He feels that phrenicectomy should not be done in pulmonary tuberculosis with cavities, when a pneumothorax is possible, when a total or partial thoracoplasty can be done, or when a tamponing can be performed.

Hudson, B., and Haeberin, F.: (*Lancet*, 1: 1115 [May 27] 1933) report on fifty cases of extrapleural plombage. They advise it in those patients with fibrocavernous type of disease localized in the apex, with the general health still good and when severe interference such as thoracoplasty is not warranted. They found that the operation may be done with safety in bilateral disease. In the fifty cases in this series there has not been any suppuration, perforation of the lung, extrusion of the paraffine or other reactions except occasional mild fever.

Slavin, P.: (*Am. Rev. Tuberc.* 29: 629 [June] 1934) finds that phrenicectomy as an aid in closure of pulmonary tuberculous cavities presents the least radical procedure, and its application in selected cases saves the patient the inconvenience of prolonged pneumothorax treatment, the risk of major surgical-collapse operations and the uncertainty of protracted expectant therapy.

B. C. G.

Calmette, A.: (*Brit. J. Tuberc.* 27: 1 [Jan.] 1933) sent out a questionnaire to 280 doctors who had used B. C. G. in children of their own families. All of the 280 doctors considered B. C. G. harmless.

Feldman, W. H.: (*Ann. J. Path.* 8: 755 [Nov.] 1932) feels that the B. C. G. is not devoid of pathogenicity for guinea pigs, and that the assertion, therefore, that the organism is innocuous can not be accepted without reservations.

Calmette, A.: (*Lancet*, 1: 653 [March 25] 1933) replies to the English Ministry of Health regarding their objection to the use of B. C. G. vaccination. The Ministry gave the following arguments against its use:

(1) Tuberculosis mortality in young children is decreasing steadily in Great Britain and this decrease has been caused exclusively by measures of hygiene, isolation of contagious patients, and improved social conditions.

(2) It is difficult and often impossible to separate vaccinated babies from their contaminated families, athough this is the essential condition of success in B. C. G. vaccination.

(3) It is not yet established how long a child vaccinated with B. C. G. may resist subsequent virulent infections.

(4) It is feared that the introduction of B. C. G. would impair the efficiency of usual measures of prophylaxis in which the governmental and local authorities have the greatest trust. Calmette replied as follows: While the death rate from all causes during the first year of life may have decreased, there has been no notable decrease of the death-rate from tuberculosis between the ages of fifteen and forty years either in England or in other countries of Northern Europe, while the number of persons giving a positive tuberculin reaction is probably the same to-day as it was twenty years ago. Tuberculosis can not be eradicated by hygienic measures alone. Only the presence in the human body of living tubercle bacilli renders it immune to tuberculosis. B. C. G. requires five weeks for the elaboration of immunity and usually segregation of the vaccinated child can be arranged for this length of time. To maintain tuberculin-allergy through puberty, revaccination may be

done every four or five years. He finally adds that the introduction of B. C. G. vaccination would not impair the efficiency of the usual measures of prophylaxis, but would rather encourage such measures.

(We do not feel that the late Dr. Calmette has satisfactorily answered the objections of the British Ministry of Health. J. B. H.-M. J. S.)

PNEUMONOCONIOSIS

Kilgore, E. S.: (*J. A. M. A.* 99:1414 [Oct. 22] 1932) reports six cases of acute pneumonoconiosis due to scouring powder. The patients were employed in an industry manufacturing scouring powder for domestic use. In four cases symptoms started from nine to twenty-four months after the beginning of the exposure and only one of the six patients remained alive, death having occurred in five instances in periods of fourteen, twenty-one, thirty-three, fifty and eighty-four months after the employment and subsequent exposure began.

Russell, A. E.: (*Am. J. Med. Sc.* 185:330 [March] 1933) investigated the effect of cement dust upon workers. Of 570 workers examined, only 3.7 per cent were diagnosed as having either positive or suspected pulmonary tuberculosis. In only two cases was the disease active at the time of the first series of special chest examinations, and neither appeared to progress as a result of exposure dusts. The x-ray films of all the cement workers contained evidence of more calcifications than those in the series made on granite cutters.

Gardner, L. U.: (*Am. Rev. Tuberc.* 29:1 [Jan.] 1934) is of the opinion that the combination of silicosis and tuberculosis is a new condition and not merely a combination or a superimposition of one process upon the other. He states that no other known dust creates a specific susceptibility to such infection except possibly silicon carbide. He adds that at least 75 per cent of those human beings who develop silicosis die of tuberculosis.

Lanza, A. J. and Vane, R. J.: (*Am. Rev. Tuberc.* 29:8 [Jan.] 1932) studied the prevalence of silicosis in the general population and its effects upon the incidence of tuberculosis. They feel that in the United States the number of industrial workers exposed to silica dust hazard is in excess of half a million. The mortality from tuberculosis and the extent of family infection is therefore a very serious problem in this wage-earning group.

Cummins, S. L.: (*Am. Rev. Tuberc.* 29:17 [Jan.] 1934) points out that both in gold miners and in coal miners there is a long-drawn out phase of pneumonoconiosis during which the gradually increasing lung fibrosis and lymph stasis still remains below the "clinical threshold" and the individual continues able to work. He found, however, that among coal miners the "miner's dyspnea" shows but little tendency to develop into "miner's phthisis". He feels that our knowledge of the etiology of pneumonoconiosis is still too imperfect to justify any sharp differentiation of "silicosis" and "silicatosis" and the condition known to-day as "silicosis" may yet be found to be due in whole or in part to a silicate.

Pancoast, H. K., and Pendergrass, E. P.: (*Amer. Rev. Tuberc.* 29:43 [Jan.] 1934) emphasize the fact that superimposed tuberculosis is a most important factor in the incapacity and mortality resulting from silicosis and other industrial forms of pneumonoconiosis. The usual clinical manifestations of tuberculosis are often characteristically lacking. They especially point out that the roentgenological study is the most dependable means of diagnosis under such circumstances. The great necessity for care as to the hygienic surroundings in both working and living quarters of individuals exposed to silica and other possible harmful dusts is stressed in their discussion.

TUBERCULOSIS AND DIABETES

Kramer, L. I. and Lawson, H. A.: (*Ann. Int. Med.* 6:1426 [May] 1932-33) reviewing their work at the Rhode Island General Hospital as to the incidence of diabetes and tuberculosis conclude that it is no greater in diabetics than in non-diabetics. They encountered no case of the bronchopneumonic type as reported by Sosman and Steidl.

Root, H. F.: (*New Eng. J. Med.* 210:1, 78, 127, 192 [Jan.] 1934) in a very thorough study at the George F. Baker Clinic, Boston, takes up the epidemiology, pathology, clinical features and treatment of diabetes associated with tuberculosis. He finds that the incidence of active tuberculosis in 1121 diabetic autopsies was 28.4 per cent and in 51,705 non-diabetic autopsies was 22.9 per cent. Tuberculous infection in diabetic children was more than thirteen times as frequent as among Massachusetts school children. Among adolescent diabetics between the ages of 15.1 and 19.9 years, pulmonary tuberculosis occurred sixteen times as frequently as among Massachusetts high school students. Active pulmonary tuberculosis was found in 2.8 per cent of the diabetic adults. Tardy diagnosis seems to be the rule. Only ten cases were discovered with a minimal pulmonary lesion. Onset of tuberculosis was subsequent to onset of diabetes. Roentgenograms showed multiple areas of calcification indicating former infection. Moderate and advanced processes formed 89 per cent of the tuberculous cases. No special diabetic types of lesions were observed, although exudative pneumonic types with cavitation frequently developed in patients with evidences of previous infection. The incidence of pulmonary tuberculosis increases with the duration of the diabetes. In forty-nine cases tuber-

culosis developed after the age of sixty years. As to the treatment, in order to promote good nutrition and resistance to tuberculosis, he advises the use of insulin almost immediately in all youthful diabetics. Sixty-nine fatal cases of tuberculosis and diabetes treated with insulin lived 8.6 years, whereas ninety fatal cases treated without insulin lived only 5.4 years after onset of diabetes. Prognosis for the tuberculous diabetic depends upon earlier diagnosis of the tuberculosis by more frequent physical and x-ray examination. The author stresses the advantages of sanatorium treatment and the use of collapse therapy in conjunction with the diabetic régime. He feels that the modern prolongation of life of the diabetic and his greater entrance into general activities expose him to tuberculosis and may, therefore, result in a greater incidence of tuberculosis among diabetics.

MISCELLANEOUS

Greengard, J. and Abrams, I. R.: (*Am. Rev. Tuberc.* 28:236 [Aug.] 1933) report two cases of spontaneous tuberculous pneumothorax in infancy. Such a pathological condition is rare in this age group, since rapidly progressive tuberculous lesions in the lung in the nature of a primary infection tend to be complicated by marked early generalization into a miliary tuberculosis. In these two cases the onset was insidious. From a prognostic point of view they feel that tuberculous pneumothorax in infancy is to be looked upon as a manifestation of a malignant type of infection.

Opie, E. L. and McPhedran, F. M.: (*Arch. Int. Med.* 50:945 [Dec.] 1932) studied the incidence of tuberculous infection in husbands and wives with a tuberculous consort. The frequency of infection in wives exposed to husbands with tubercle bacilli in the sputum was 35.5 per cent; in those exposed to husbands with no demonstrable tubercle bacilli 22.9 per cent. The incidence of infection to husbands exposed to wives with open tuberculosis was 45.6 per cent and when there were no tubercle bacilli in the sputum it was 35.9 per cent. Latent apical tuberculosis, like clinically manifest tuberculosis, was more frequent in husbands than in wives.

Martin, D. C.; Pessar, H. T., and Goldberg, J. A.: (*Am. Rev. Tuberc.* 29:182 [Feb.] 1934) report on their tuberculosis survey among 2000 food handlers in New York City. They found active tuberculous pulmonary lesions to the extent of approximately 2 per cent in apparently healthy food handlers. In addition they found evidence in many of arrested tuberculosis, latent childhood tuberculosis and pleurisy. They are outspoken concerning the comparative value of physical examination and x-ray, stating that when the question of tuberculosis is involved, no physical examination of the chest is of any value without the x-ray.

(This extreme statement is distinctly open to question. J. B. H.-M. J. S.)

Watts, H. F. R.: (*Am. Rev. Tuberc.* 29:424 [April] 1934) gives his observations of the x-ray examination in the diagnostic clinics of the Boston Health Department. Of 10,639 x-ray examinations 5,023 were adults and 5,616 were children. In the adult group 7½ per cent showed roentgen evidence of childhood type of tuberculosis uncomplicated by phthisis, 22½ per cent showed roentgen evidence of phthisis without evidence of the childhood type; while 2 per cent had both types of the disease. In the group of children 9 per cent were found to have childhood type lesions unaccompanied by adult phthisis. Sixteen children had adult phthisis unaccompanied by the childhood type, while twelve had both childhood and adult types. The author notes especially that of the ninety-two adults having evidence of both childhood and adult types, the adult lesions were frequently moderate or minimal in extent and showed evidence of fibrosis and healing.

MEDICAL PAGEANTRY

The medical pageants presented by the students of Tufts College Medical School, under the direction of Dr. B. Spector, Professor of Anatomy, have found a definite place in the fostering and teaching of medical history. They furnish something tangible to arouse the interest of the student and their visual character appeals to the busy physician. Professor Henry E. Sigerist, Director of the Institute of the History of Medicine at Johns Hopkins University, and Dr. Charles Singer, of London, have been impressed with their value in the teaching of the history of medicine when the pageants are produced in a serious manner.

They have accomplished much in the way of interesting the students in the history of their chosen profession and have become so popular that there are enough candidates to put on three pageants instead of the single one which it is possible to produce annually.

These pageants were started in 1929 and have been produced every year and a special one on surgery was given for the American College of Surgeons during the convention of last month.

The public performance of 1934 will be given at the Boston Medical Library on the evening of November 19 at 8 o'clock under the auspices of the Boston Medical History Club. It is to be devoted to the fundamental concept of the evolution of circulation and respiration. In addition, the history of midwifery will be portrayed by women students of the school.

The students write their own parts, after research into the writings of the scientists whom they are to represent, which are edited by the Director. The characters are costumed in the correct dress of their periods.

In connection with the pageant, there will be an exhibition of the texts of the characters represented, selected from the material in the Boston Medical Library and described by Mr. Ballard, Director of the Library.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 20461

PRESENTATION OF CASE

Approximately six months before entry the patient, a sixty-five year old Canadian motorman, noticed increasing weakness and shortness of breath. While working in cold weather he developed numbness of his hands, but there was no tingling or paresthesia. In a period of one month these symptoms had become so severe that he was forced to stop work. His shortness of breath was very severe after climbing stairs and, recently, even while walking. His ankles became swollen, especially at night. His skin became slightly yellow and sallow. There was no orthopnea, cough or pain. He did have severe palpitation on slight exertion. His appetite was slightly decreased and he became definitely constipated. He began to vomit almost daily. The vomiting was preceded by nausea but was not related to meals. The vomitus consisted of mucus and bile and was never blood-streaked. There was no abdominal pain, postprandial distress or melena. A physician told him he was run down, that his blood was poor, and put him on iron. Four months before entry he had improved sufficiently so that he returned to work. He was still forced, however, to take cathartics for his constipation although he had stopped taking iron. His complexion remained sallow. One month before entry his weakness and shortness of breath returned. He began to vomit again and developed a very annoying soreness of his tongue, making it impossible for him to drink hot tea or to eat sour foods. He developed a distressing sensation in the midsternal region about one-half hour after meals and lasting about an hour. It felt as if the food were blocked there. He had no difficulty in swallowing, however. Three weeks before admission he was forced to give up work because of weakness and shortness of breath. Two weeks before admission he was seen in the out-patient department, and admission to the house was advised.

The family and marital histories are non-contributory.

He was in this hospital a year before his present entry for reduction of an incarcerated hernia and a Bassini repair. No blood studies other than a white cell count were done. The white cell count was 21,000 on admission and 6,700 on discharge.

Physical examination showed a fairly well-developed elderly man with a slightly icteric tint to his skin. The lungs showed diminished breath sounds and occasional moist râles at the left base. The heart was slightly enlarged and there was a soft blowing apical systolic murmur heard loudest in the sixth space in the anterior axillary line. The blood pressure was 110/70. Abdominal examination showed no visible peristalsis. There was slight tenderness in the midepigastrium. No masses were palpable. Rectal examination was negative.

The temperature was 99.5°, the pulse 95. The respirations were 24.

Laboratory examination of the urine was negative. The blood showed a red cell count of 1,380,000, with a hemoglobin of 45 per cent. The white cell count was 1,600, 57 per cent polymorphonuclears. A smear showed marked anisocytosis and moderate poikilocytosis. There were many macrocytes and microcytes. The platelets were reduced in number. A gastric analysis showed no free hydrochloric acid after test meal and after histamine. Examination of the stools was negative. A Hinton test was negative. The non-protein nitrogen of the blood was 34 milligrams.

A gastro-intestinal series done in the out-patient department showed a normal esophagus. There was an irregular filling defect, showing evidence of craters involving the lower half of the stomach. The duodenum showed a constant deformity which appeared to be extrinsic. A reëxamination after admission to the house confirmed the previous observations.

Gastroscopy showed that the entire mucosa was very pale and thin so that numerous small blood vessels could be seen very well. Normal rugae were almost entirely absent. On the lesser curvature and posterior wall in the antrum of the stomach there was a rounded, elevated, red lesion which was fairly smooth in contour and which was attached to the mucous membrane by a broad base. This lesion was about seven or eight centimeters in diameter. The mucosa was typical of the atrophy seen in pernicious anemia.

He was transferred to the Medical Service where he was treated with liver extract and transfusions. Before treatment his red blood cell count was 1,300,000; reticulocytes 1.8 per cent; oxygen capacity 6.4 volumes per cent; volume of cells 15.8 per cent; volume index 1.42 and color index 1.34. After three weeks of treatment the red cell count reached 1,880,000; reticulocytes 15.2 per cent; oxygen capacity 10.5 volumes per cent; volume cells 24.4 per cent.

Approximately one month after entry operation was performed.

Differential Diagnosis

Dr. Arlie V. Bock: Increasing weakness and shortness of breath are two leading symptoms which make me think first of all of anemia. The other outstanding things one might think of would be a cardiac syndrome, or the question, possibly, of some advanced intrapulmonary or intrathoracic tumor, or a parenchymal lesion of the lung.

After a month his symptoms were so severe that he was forced to stop work. He has a fairly rapidly progressive condition.

"His ankles became swollen, especially at night." That finding of course could appear in a cardiac state or severe anemia; or there is the other possibility that I might have mentioned, chronic nephritis, which might be responsible for the symptoms so far mentioned.

The fact that his skin was slightly yellow and sallow leads more to the idea of anemia in the background of the condition.

"There was no orthopnea, cough or pain." That goes a long way to rule out heart disease. He had severe palpitation on slight exertion, which we might expect if the heart were at the bottom of it, but he would not be able to lie flat. Slight palpitation on exertion is a common finding in anemia.

The daily vomiting is the first intimation of a possible gastric lesion, although it is often a terminal stage in chronic nephritis.

"The vomitus consisted of mucus and bile and was never blood-streaked." Apparently some of this material was regurgitated through the pylorus of the stomach.

We would like to know how much iron and what sort of iron he was given. Apparently he got some relief from the iron, which is unusual, in view of the subsequent notes.

Soreness of the tongue of course is one of the characteristic parts of the history of most cases of pernicious anemia.

"He developed a distressing sensation in the midsternal region about one-half hour after meals and lasting about an hour." That is the first history of discomfort, localized, and having its origin presumably in the stomach.

"He was forced to give up work because of weakness and shortness of breath." That makes one think strongly of the possibility of pernicious anemia in the background at the age of sixty-five, rather than heart disease, or a condition like chronic nephritis, because it is astonishing how long a patient with pernicious anemia is able to stay at his work before giving up.

The only other hospital connection is that he was in the hospital a year previously for repair of a hernia, at which time we have only the data on a white count. It would be quite interesting had a blood smear been done at that time.

On physical examination he has a slightly icteric tint to his skin, which is again characteristic of the finding in pernicious anemia.

The murmur described in the heart is presumably a hemic murmur.

If the figures of the blood examination are true, the diagnosis of pernicious anemia is difficult to escape. I should like to know if there were other white blood cell counts done in this case because it is a somewhat unusual finding. Pernicious anemia is more apt to have five, six, or seven thousand white cells unless the patients are so ill they are in an aplastic state, and then we should not expect fifty per cent polymorphonuclears in the blood smear. If this patient were having hemorrhage, one would expect a higher count than that. That is a queer finding in this account of the blood. The blood smear is quite characteristic of pernicious anemia, with macrocytes, microcytes and reduction of blood platelets.

The gastric analysis is characteristic; it was done after histamine.

The non-protein nitrogen rules out the question raised about the possibility of chronic nephritis of a grade sufficient to produce the anemia that he has.

It is interesting that the reëxamination of the gastro-intestinal tract shows the same findings. With this description, when the x-ray people talk about visible craters, it is hard to escape the feeling that one is probably dealing with a cancer of the stomach.

Dr. Tracy B. Mallory: Dr. Hampton will demonstrate the x-rays.

Dr. Bock: I might just say that this other note about the constant deformity of the duodenum which appeared to be extrinsic might possibly be occasioned by a gall bladder full of stones or a chronic cholecystitis with inflammation and adhesions.

X-ray Interpretation

Dr. Aubrey O. Hampton: I had to select these films for lantern slides so I know too much about the case to come to any definite conclusions without bias; but as I saw this film first I noticed this filling defect here which is certainly in the stomach, intraluminal, and which seems to swing around in this direction. Here we have an irregular lesser curvature which looks very much like an ulcer crater. On that film alone we would suspect very strongly a carcinoma occupying this area with the margin going around here and fading out because of lack of pressure or covering up by too much barium. The duodenum is large and smooth, and here is the pressure defect described. That

is usually interpreted as a pressure defect of the gall bladder.

On this film we have a different appearance. We still have the defect in the stomach extending from the lesser curvature and in this film a complete circle in the area suspected as a crater. We would say that this is the margin of a large ulcer but for the defect in the duodenum, where there is a round filling defect which is everywhere surrounded with barium and does not appear to be attached except in this area near the pyloric valve, where there is a filling defect.

Differential Diagnosis Continued

Dr. Bock: Dr. Hampton has succeeded in making it a little more difficult than I supposed from the description here, but there is a helpful note in the next paragraph about the gastroscopy, which shows pretty complete atrophy of the mucous membrane of the stomach with absence of normal rugae and the presence of a definite lesion on the lesser curvature posteriorly in the antrum of the stomach. The lesion measures seven or eight centimeters. That is a much larger lesion than we usually think of in connection with simple ulcer; but I do not know enough about the extrinsic appearance of malignant disease to know whether this suggests malignancy to Dr. Benedict, who presumably did the gastroscopy.

The patient was transferred to the Medical Service for further observation, and these additional findings about the blood make the diagnosis of pernicious anemia seem certain. Before treatment the red count was 1,300,000, the reticulocytes 1.8 per cent, the oxygen capacity 6.4 volumes per cent, which is compared with the normal of 18 to 20. The volume of cells was 15.8, against 44 or 46. The volume index was 1.42, which is high. It only reaches that figure apparently in pernicious anemia. As a rule, the color index is high, the two of course going together.

The next statement is undoubtedly misleading. He could not have had three weeks' treatment of pernicious anemia in this hospital and have a red cell count of 1.800,000 unless there was something peculiar in his response. We should expect at the end of three weeks a much higher red cell count than that. I should guess that the first extract used was not a very potent one and that after a shift of extract he got a very characteristic but moderate response of reticulocytes, 15 per cent. The oxygen capacity rose to 10.5, and the volume of cells to 24.4.

I believe there is no doubt about the diagnosis of pernicious anemia. The only question in my mind is what the lesion in the stomach might be. In thinking about this case it is obviously not so simple as it looks for this reason. We have in this clinic, and in other clinics of this size, relatively large numbers of cases of cancer of the stomach, and we have seldom found pernicious anemia associated with cancer of the stomach. Dr. Jacobson tells me that in sixty-five carefully studied cases of pernicious anemia in the past two years no case of cancer of the stomach has been found in association with this disease. Such experiences make me skeptical as to whether we are dealing with cancer of the stomach. I believe that the man has pernicious anemia and whatever he has in his stomach is an independent condition. It is hardly fair to assume that he has cancer of the stomach, and then gastritis and atrophy of the stomach, and then pernicious anemia, for the simple reason that the great majority of cases of cancer of the stomach are seldom associated with pernicious anemia. This interests me a good deal because back in my houseofficer days, in 1916 and 1917, we were then x-raying the cases of pernicious anemia, of which there were always a great many more in the hospital than at the present time,—it was not at all unusual to have six or seven cases on each medical service at a time—and for some reason we had the stomachs of these patients x-rayed to see if they had carcinoma, but I do not remember finding any. It is interesting to speculate as to why we do not have more cases of pernicious anemia in connection with cases of cancer of the stomach because these lesions are chronic ones, often running a long period of time. There is a very high percentage of cases associated with achlorhydria, even after histamine, indicating change in the function of the mucous membrane of the stomach which is the seat of the "x" factor supposed to be responsible for the care of the blood-forming machinery in this disease, and, according to Castle's work, being the cause of pernicious anemia.

I will make a diagnosis of pernicious anemia and leave the question open to doubt as to whether this is cancer, merely on the very rare incidence of cancer in association with pernicious anemia. I believe if this is cancer it is an independent finding in this patient.

Clinical Discussion

Dr. Mallory: Dr. Benedict, you looked into this man's stomach. Will you describe the findings?

Dr. Edward B. Benedict: I should like to say a word about the gastroscope. We have been using the Wolf-Schindler flexible gastroscope for a year and a half, and are finding it very valuable. Because of the flexibility of the lower end of the instrument it is an easy and safe examination, and is carried on under local anesthesia. We have examined one hundred and fifty cases.

I will show you lantern slides of the stomach as seen by gastroscopy. In the first slide one sees a smooth, elongated tumor which one would have no hesitation in saying was a benign polyp

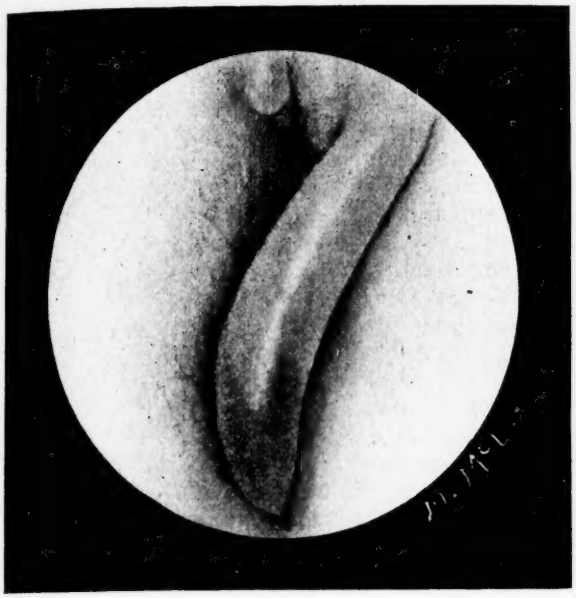

FIG. 1. Posterior wall. Appearance of polyp as seen through the gastroscope.

of the stomach (fig. 1). However, looking at the base of the tumor in the next slide it seems to have a broad attachment and suggests malignancy. Dr. Allen and I looked up a series of polyps of the stomach and found that about forty per cent become malignant and about thirty-five per cent are associated with pernicious anemia. The third slide shows that the rest of the stomach presents a very atrophic mucosa characteristic of pernicious anemia (fig. 2). It is only in marked atrophy that the blood vessels stand out the way they do in this field. From my examination of this patient I felt that he had pernicious anemia associated with a gastric polyp, which was possibly undergoing malignant change at its base.

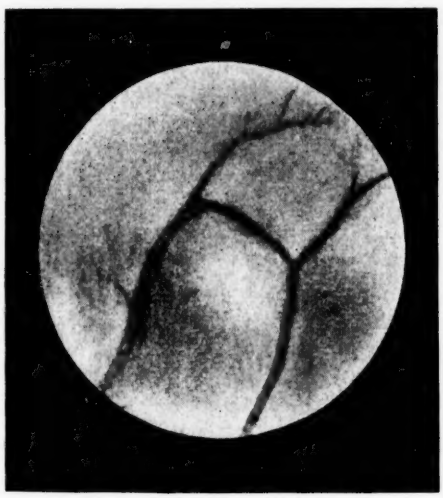

FIG. 2. Greater curvature. Atrophic appearance of mucosa typical of pernicious anemia (gastroscopic drawing).

DR. MALLORY: Dr. Hampton, have you any more to add?

DR. HAMPTON: I think there is an interesting comparison between the size of the rugae here by x-ray and gastroscopy. The rugae we saw were very large. We would call them hypertrophic rugae. Dr. Benedict says they were atrophic. We rely on Dr. Benedict and his gastroscopic findings for the final opinion on the mucosal changes in the stomach.

There is one differential point here that I did not mention because I couldn't very well, and that is that this could not be the margin of the ulcer because mucosal folds cross it. Since I know what it is, now may I say what I know?

DR. MALLORY: Yes.

DR. HAMPTON: This is an elongated polyp attached up here in the fundus and dangling through the pyloric valve into the duodenum. I have never seen one like it before.

PATHOLOGIC DISCUSSION

DR. MALLORY: As Dr. Hampton has told you, the lesion that was found at operation was a large polyp attached at the fundus of the stomach and running down the entire length of the stomach, eventually passing into the duodenum. We have a picture of the specimen here. You can see how long and slender it was.

Pernicious anemia has been shown within the last few years to have a very direct bearing on gastric pathology. Last year Dr. Madelaine Brown* looked up all the cases of pernicious anemia that had come to autopsy at the Boston City, the Peter Bent Brigham, and this hospital. She got 150 cases, and a really normal stomach was found in only one case. The majority of the cases showed little except an atrophic mucous membrane or a severe gastritis; in either case with more or less complete loss of the acidophilic chief cells which presumably secrete the acid. In a certain number of cases, more significant gastric pathology was present. As Dr. Bock brought out, the association with cancer is not very frequent. She found only one cancer. On the other hand ten benign gastric polyps were present in the series; about seven per cent. My personal experience is that gastric polyps are much commoner than that, and in the cases in the past five years we have had about a thirty per cent incidence of benign polyps. Dr. Brown's figures are based on autopsies going back to 1890, a large proportion of which came from the period before pernicious anemia was treated with liver, so it is probably fair to assume that the duration of life was shorter than cases coming to autopsy now. The fact that we are seeing gastric polyps more frequently now

*New Eng. J. Med. 210:473, 1934.

suggests that the longer duration of disease since the introduction of liver therapy allows time for them to develop. I believe that we are going to see many more cases of gastric polyp associated with pernicious anemia than have been observed in the past.

Have you any further comment, Dr. Bock?

DR. BOCK: I meant to say something about polyps being the one lesion that is apt to be found in the stomach in pernicious anemia.

DR. MALLORY: Personally I know that I have seen several cases of cancer of the stomach associated with pernicious anemia, more than those statistics would suggest.

A PHYSICIAN: Was there any record of further white cell counts?

DR. MALLORY: There were two white cell counts, both below 2,000.

CASE 20462

PRESENTATION OF CASE

A fifty-two year old American machinist entered complaining of weakness associated with fever of two weeks' duration.

Two weeks before entry, while returning to work after lunch, he became very hot, which he attributed to the shop in which he was working. He kept on working until he felt somewhat "all in" and "burnt up". His skin was cold and he felt somewhat weak and shaky, although he had no chills, flushing or dizziness. He left work that afternoon and walked home, a distance of about one mile. How he reached home and the various events that occurred between two and six o'clock that afternoon were extremely hazy. He went to bed that evening without eating any supper. There was no cough or pain. He was seen by a physician who said that he probably had typhoid fever. His temperature ranged between 100° and 105°. On one occasion during the following week he developed a shaking spell of about ten minutes' duration. Since then he had two or three similar attacks. These were not followed by excessive perspiration. The last chill occurred while in the Emergency Ward on the day of admission. His appetite was poor. His stools were loose but not clay-colored or tarry. There was no jaundice. He had been confined to bed during this two-week period. There was no jaundice. There was no history of a previous similar attack or of anybody else in his neighborhood having a similar condition.

The family, marital and past histories are non-contributory.

Physical examination showed a well-developed and fairly well-nourished man showing some evidence of loss of weight. His skin was ashy gray and moist. The tongue was dry, coated and red. There was some tremor of the fingers. The heart was normal in size. The sounds were of good quality and regular. The first sound at the apex was doubled. There was a soft systolic murmur. The spleen was not felt. The liver was felt three to four finger-breadths below the right costal margin. There were no rose spots.

The temperature was 103.5°, the pulse 110. The respirations were 24.

Examination of the urine showed a specific gravity of 1.020. A slight trace of albumin and an occasional white blood cell and cast were noticed. Examination of the blood showed a red cell count of 5,080,000 with a hemoglobin of 95 per cent and a white cell count of 22,400 with 89 per cent polymorphonuclears. The smear was not remarkable. The stools were negative. A Hinton test was negative. Widal, paratyphoid and undulant fever agglutinations were negative. A blood culture showed type I pneumococcus. A portable film of the chest was underexposed and no diagnosis could be made.

He continued to run a very high fever—his temperature reaching as high as 106.4° and, following alcohol sponges, going down as low as 101°. On the fourth day he developed increased breath sounds and tactile fremitus at the left base and some inspiratory râles. He was given antipneumococcus type I serum with very little effect. Severe chills followed and the serum was stopped. He was given quinine hydrochloride by mouth. On the tenth day a pericardial friction rub was heard in the fourth intercostal space near the sternum. This rub was not present two days later, but reappeared that evening. He continued to go downhill very rapidly and died on the thirteenth day.

DIFFERENTIAL DIAGNOSIS

DR. WILLIAM D. SMITH: To reduce the history to the lowest terms, here is a man of fifty-two with sudden onset of fever and chills without localizing symptoms. The temperature range from 100° to 105° suggests sepsis rather than tuberculosis or typhoid. There is no evidence of other conditions that may give prolonged fever without localizing symptoms, such as Hodgkin's, bacillus abortus, malignancy, or visceral syphilis, and so on. So he simply had fever of the septic type and chills without localizing symptoms, fever.

He is sick enough to have an ashy gray skin. Someone reading on might say that is the appearance of pyemia or septicemia.

I suppose any condition which prevents the synchronous closure of the mitral and tricuspid valves may give a double first sound. We may get it in mitral stenosis, though we usually do not, and impairment of the conduction system may give a split first sound at the apex. However, that does not help any more than the presence of a soft systolic murmur helps.

The spleen was not enlarged. I wish it had been. I have a notion that he was extremely

septic, and I wish it had been felt. The liver is said to be enlarged with the edge palpable three or four fingers below the costal border; why? Certainly there is no evidence that there was a chronic passive congestion; no evidence of anything above the diaphragm, or under it. There may be something under it, although there is nothing in the physical examination suggesting it. Of all the causes of enlargement of the liver I can only think of one or two that could fit with this septic temperature and chills. He might have had an abscess or a cholangitis, infectious or purulent cholangitis; but we have no picture of tenderness, which we certainly should expect, and we have no jaundice. Of course a man can have cholangitis without jaundice because jaundice with cholangitis might depend much more on the antecedent disease, on the cause of the cholangitis rather than the cholangitis itself. Another possibility is, with the liver three fingerbreadths down, the enlarged liver may be another and an independent condition with nothing to do with the present situation. He may have had cirrhosis for a long time and it may have no relation to the present illness.

The respiratory rate of twenty-four is against lobar pneumonia.

It is queer that he should have such a good blood examination as that, with chills and fever for two weeks and high temperature, unless he was desiccated; and why should he be desiccated? There is nothing about vomiting or diarrhea. Anyhow, the white count helps us rule out typhoid and helps us rule out malaria.

"A blood culture showed type I pneumococcus." We have that to hang on to. We frequently have a pneumococcus bacteremia in severe cases of lobar pneumonia and we usually think of the pyogenic qualities of the pneumococcus in complications of pneumonia; such as peritonitis, endocarditis, meningitis, and arthritis. But there are other possible portals of entry of the pneumococcus besides the lungs. One may and does get from time to time pneumococcus septicemia from an ear, from sinuses, from the throat, or from the bile ducts. I think though that those cryptogenic, if one wants to use that word, pneumococcus infections are more common in young people than in a man in the fifties. I may be wrong about that.

Is it possible that this man had a lobar pneumonia, a central pneumonia, and then on the sixteenth day it came to light on the surface? It seems highly improbable to me. I may not think it impossible, but it is too improbable for me to consider seriously.

"On the fourth day he developed increased breath sounds and tactile fremitus at the left base and some inspiratory râles." I should like to know if that amounted to anything; whether he developed frank pneumonia, or whether he developed empyema. There are no further comments on the lungs. We are left up in the air. Reading further one wonders whether that might have been due to a pericardial effusion, Ewart's sign, but the only definite information we have is that he had changes at the left base.

"A pericardial friction rub appeared and disappeared after two days." The fact that the pericardial friction rub disappeared after two days makes us think he might have developed fluid; then they spoil it by saying it reappeared that evening. Yet that pericardial friction rub raises the question. Is that pericarditis part of the underlying disease or is it a terminal infection?

DR. TRACY B. MALLORY: Perhaps Dr. Holmes can help us.

X-RAY INTERPRETATION

DR. GEORGE W. HOLMES: The films that we have are portable films taken nine days before the patient died. The heart shadow is normal in shape but does appear large. Part of that is due to the way the films were taken, but I think there is some actual increase in the size of the heart. There is nothing to suggest a pericardial effusion or any other definite lesion. The diaphragm is normal on both sides. The question was raised as to enlargement or displacement of the liver. I do not think we have any enlargement. The diaphragm is normal on both sides. In the lower part of the right lung the aëration is not quite so good as it is on the other side, but certainly the changes are not enough to represent consolidation. I should think that all the changes we see might very well be circulatory. There is no evidence of pneumonia, no evidence of subdiaphragmatic abscess, and no evidence of pericardial effusion.

DIFFERENTIAL DIAGNOSIS CONTINUED

DR. SMITH: It is a little difficult for me to figure out whether that plate was taken before they found the tactile fremitus or later.

I am going to assume that the man who heard the pericardial friction rub knew what he heard.

Beyond the fact that this man was apparently septic and very toxic it seems to me we have only one diagnostic lead. We have a positive blood culture of pneumococcus and we have the story of pericarditis. I am going to jump into the lead because if that is not right, I have not even an intelligent guess. I will say that he had pneumococcus septicemia, that he had pneumococcus pericarditis; and then I will hazard this further opinion and say that he also had pneumococcus endocarditis. Possibly Dr. Mallory will show other local areas of infection, possibly suppuration in the liver, to account for the big liver.

If it is not out of order, I should like to ask if someone has a good bet.

DR. MALLORY: We always welcome alternative diagnoses.

Dr. Albright, do you want to tell us your diagnosis?

DR. FULLER ALBRIGHT: I left the service before this patient died. I do not remember the details very well. The diagnoses on the record at death were pneumococcus type I septicemia; pericarditis?; bronchopneumonia, terminal. On looking over the record I find that only one examiner felt the liver. I, personally, did not.

DR. MALLORY: I think it would be very difficult to arrive at any other diagnosis on this case, and yet it is not entirely correct.

CLINICAL DIAGNOSES

Septicemia (pneumococcus type I).
Bronchopneumonia.
Pericarditis.

DR. SMITH'S DIAGNOSES

Pneumococcus septicemia.
Pneumococcus pericarditis.
Pneumococcus endocarditis.

ANATOMIC DIAGNOSES

Endocarditis, acute bacterial, tricuspid valve, pneumococcus type I.
Pulmonary thrombosis and infarction.
Septicemia, pneumococcus type I.
Multiple petechiae and suggillations of skin.
Septic arthritis, right sternoclavicular joint, pneumococcus type I.

PATHOLOGIC DISCUSSION

DR. MALLORY: It is quite true that the patient did have pneumococcus endocarditis, but he did not have any trace of pericarditis. The endocarditis was a most unusual one. I think that even from your seats you can see the vegetation on the tricuspid valve; it is somewhat larger than a golf ball and completely fills the right ventricle, the largest acute vegetation I have ever seen. The valves on the left side were entirely negative, and his endocarditis being on the right of course the absence of multiple petechiae is explained. The emboli were in his lung, which was riddled with infarcts, but no emboli in the peripheral circulation whatever. I think it is another example of the infrequency with which the x-ray picks up infarcts, in contrast to the regularity with which it picks up pneumonia. It is something I have never felt able to explain at all.

DR. SMITH: I think the person who reported the pericardial friction ought to explain.

DR. ALBRIGHT: Fortunately it was reported after I left the service.

DR. MALLORY: I have no very clear explanation of the murmurs, but it seems to me that so enormous a vegetation as that would give strange murmurs. It was slightly pedunculated and served almost as a ball-valve in the tricuspid valve.

A PHYSICIAN: With such a condition as that, would there not be a pulsation visible in the right auricle?

DR. MALLORY: He had no evidence of heart failure whatever. There was no passive congestion. The death was definitely a septic one.

Dr. White, have you an opinion?

DR. PAUL D. WHITE: Was there no evidence of underlying pathology in the tricuspid valve, either rheumatic or congenital?

DR. MALLORY: No.

DR. WHITE: The other point I have in mind is that pneumococcus endocarditis was well described a year or two ago by Dr. Lord before the New England Heart Association. Do you remember the date of your paper, Dr. Lord?

DR. FREDERICK T. LORD: It was published in the *New England Journal of Medicine* (Vol. 207, No. 18, p. 767, Nov. 2, 1932).

DR. MALLORY: Do you remember whether any of those cases had tricuspid lesions, Dr. Lord?

DR. LORD: The tricuspid valve was involved in five of twenty-three cases of pneumococcus endocarditis at the Massachusetts General Hospital.

A PHYSICIAN: Why was the liver four fingerbreadths down?

DR. MALLORY: It was at the costal border and normal in size. I meant to comment on that.

DR. HAMPTON: Were there any old infarcts in the lungs, from a week to ten days?

DR. MALLORY: They were quite variable in age. There were thrombi showing all degrees of organization throughout the pulmonary arteries.

DR. MEANS: I think pleuropericardial friction sounds are very difficult to distinguish from true pericardial friction sounds. In exophthalmic goiter we often hear a scratchy noise under the manubrium which is a good imitation of pericardial friction sounds; yet when the patient is rid of the thyrotoxicosis the sound disappears. I do not think anyone has explained how it is brought about. I do not think sounds of that kind are infrequent. The sound here may be of a similar sort.

DR. WHITE: We hear a similar to and fro friction sound in cases of pulmonary embolism with marked distention of the pulmonary artery and right ventricle; this fulness of the pulmonary artery may play a rôle in the case of the overactive thyrotoxic heart, too. We know that fluoroscopy of patients with thyrotoxicosis frequently shows unusual prominence of the pulmonary artery.

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PUBLICITY OF MEDICAL AFFAIRS

PUBLICITY of matters relating to medicine and physicians from time to time serves to focus our attention on the need for some definite plan whereby medical items of legitimate news interest may be presented to the public in accurate, simple and readable fashion without violation of the tenets of medical ethics. It must be recognized that the function of a newspaper is to disseminate items of interest and value to its readers. Editors realize (as do advertisers, all too obviously), the great interest that all people have in health and matters pertaining thereto.

Newspapers are desirous of getting and publishing accurate and news-worthy information. Accuracy of representation of scientific facts is usually as fond a desire of the press as it is of the medical profession, though at times sensationalism overcomes discretion. Traditionally the doctor has come to regard the newspaper reporter and editor as men to be shunned for fear that his comments may be distorted and misinterpreted by them in the published report of an interview. Those of us who are readers of the magazine *"Time"* realize what can be done in the way of reasonable, accurate and interesting medical reporting. Accurate and ethical presentation of medical news is by no means impossible.

The problem is not an insoluble one. One great need is an authoritative medical body equipped to give accurate and unbiased information on topics of medical interest to the press.

Reporters and editors alike are deeply interested in the success and progress of the medical profession. They are at least in part cognizant of the ethical problems presented in the utilization of names of practicing physicians and surgeons in connection with various news stories. We have all of us fumed over mistakes in the reporting of medical matters, little has been done to correct the situation and when a reporter has attempted to obtain help in preparing a story on a medical subject a really competent representative of the profession has often felt obliged to refuse to discuss a given subject.

The present lack of system is thoroughly unsatisfactory to the medical profession and to the newspapers. In controlling publicity relative to an important medical discovery, the writer was impressed by the uniform coöperation and understanding of the editors of the Boston newspapers. Would peptic ulcer sufferers have been writing, telegraphing and calling their physicians with the fear of certain gastric cancer weighing them down, had one of the reports of a recent clinical program been adequately controlled? We need not further multiply the instances.

Our instinct is to blame the press for all these inaccuracies. Instead, we should blame ourselves in large part.

If a carefully chosen committee of medical men had prepared releases for the press and given impersonal interviews explaining to the reporters the true worth of each announced fact, these regrettable occurrences would not be rankling now.

This matter of reporting of medical discoveries or medical news in the lay press is of utmost importance to the prestige of the profession. It should be a prime concern of the Massachusetts Medical Society to see that this is properly handled. It might be well at the next meeting of the Council to consider the feasibility of establishing a Publicity Committee which would be concerned with press releases, and form a liaison between the medical profession and the press, and would be a group to whom reporters and editors alike could turn for accurate information on medical matters.

Certainly we could be no worse off than we are at the present time. In an endeavor to in-

terpret scientific news the average reporter or editor is as much in need of help as is the average doctor when it comes to understanding matters of publicity and newspaper practice. In an experience of some years with Boston papers the writer has had uniformly courteous consideration and real effort has been made by the press to meet the demands of accurate and ethical medical publicity. We must coöperate with the press or have only ourselves to blame for the raising of false hopes in thousands of sufferers and ultimately, through too many false heraldings of the dawn, to see all medical discoveries viewed with skepticism.

REGIONAL CONFERENCE OF THE AMERICAN ACADEMY OF PEDIATRICS

THE tri-city pediatric groups, consisting of the New England Pediatric Society, the Philadelphia Pediatric Society and the Section of Pediatrics of the New York Academy of Medicine have for a number of years conducted an annual joint meeting, rotating among the cities represented. This year, however, the importance of the meetings, which were held on October 19 and 20, was greatly enhanced by the inclusion of the eastern regional conference of the young but flourishing American Academy of Pediatrics.

The meetings were held in New York, at the Hotel Ambassador, on Friday, the nineteenth, and at the New York Hospital and Cornell Medical Centre on the morning of Saturday the twentieth. The scientific sessions were composed of interesting and important papers and were well attended; the dinner at the Ambassador on Friday evening was a notable success, the principal speakers being Dr. Clifford G. Grulee, editor of *The American Journal of Diseases of Children* and secretary-treasurer of the Academy, and Commissioner John Rice of the New York City Department of Health.

The most important product of the conference, however, is its restatement of the aims and purposes of the Academy of Pediatrics, an organization to consolidate and unify the efforts and ideals of the members of this branch of medicine, and constantly to improve and maintain the methods and standards of the specialty. The Academy has already enrolled on its list a great majority of the prominent pediatrists of this country and Canada and has, in conjunction with the American Pediatric Society and the Pediatric Section of the American Medical Association, established a national certifying board in pediatrics.

The Academy has embarked on its career in a distinct atmosphere of integrity which, if it can be judged by its organization, seems likely to continue.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

EMERY, EDWARD S., JR. A.B., M.D. Harvard University Medical School 1920. Associate in Medicine, Peter Bent Brigham Hospital. Instructor in Medicine, Harvard Medical School. Address: 319 Longwood Avenue, Boston, Massachusetts. Associated with him is

WOSIKA, PAUL H. B.S., M.D. Northwestern University Medical School 1931. Formerly: Assistant Resident in Medicine, and Voluntary Graduate Assistant, Peter Bent Brigham Hospital. Address: 303 East Superior Street, Chicago, Illinois. Their subject is "A Clinical Study of Chronic Ulcerative Colitis." Page 907.

MURPHY, WILLIAM P. A.B., M.D. Harvard University Medical School 1922, as of 1920. Associate in Medicine, Peter Bent Brigham Hospital. Instructor in Medicine, Harvard Medical School. His subject is "Pernicious Anemia." Page 914. Address: 311 Beacon Street, Boston, Massachusetts.

MERRILL, A. S. M.D. Tufts College Medical School 1908. Formerly: Assistant Roentgenologist, Massachusetts General Hospital. Now: Roentgenologist, Elliott Hospital, Manchester, New Hampshire. His subject is "Pulmonary Tuberculosis Roentgenologically Considered." Page 916. Address: 814 Elm Street, Manchester, New Hampshire.

CROSBIE, ARTHUR H. A.B., M.D. Harvard University Medical School 1906. F.A.C.S. Genito-Urinary Surgeon, Cambridge Hospital, St. Elizabeth's Hospital, and Massachusetts Women's Hospital. Assistant Surgeon, Beth Israel Hospital. Consultant Genito-Urinary Surgeon, Boston Dispensary. His subject is "The Two-Stage Operation for Bladder Tumors." Page 920. Address: 520 Commonwealth Avenue, Boston, Massachusetts.

MILLER, D. K. A.B., M.D. Harvard University Medical School 1929. Formerly: Intern, Boston City Hospital. Now: Assistant Resident Physician, Hospital of the Rockefeller Institute for Medical Research, New York. Address: Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York City. Associated with him is

RHOADS, C. P. A.B., M.D. Harvard University Medical School 1924. Associate Member, Hospital of the Rockefeller Institute for Medical Research. Address: Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York City. Their subject is "The Presence in Egg-White and in a Rice-Polishings Concentrate Low in Vitamin $B_2(G)$ of an Anti-Pernicious-Anemia Principle." Page 921.

COTTON, FREDERIC JAY. A.B., A.M., M.D. Harvard University Medical School 1894. F.A.C.S. Consulting Surgeon, Boston City Hospital, Faulkner Hospital, Newton Hospital, New England Hospital for Women and Children, Chelsea Memorial Hospital, and Cape Cod Hospital, Hyannis. Address: 520 Commonwealth Avenue, Boston, Massachusetts. Associated with him is

MORRISON, GORDON MACKAY. A.B., M.D. Tufts College Medical School 1926. F.A.C.S. Assistant Out-Patient Surgeon, Boston City Hospital. Visiting Surgeon, Chelsea Memorial Hospital. Junior Visiting Surgeon, Newton Hospital. Consulting Surgeon at Huggins Memorial Hospital, Wolfeboro, New Hampshire, Framingham Union Hospital, and Leonard Morse Hospital, Natick. Address: 520 Commonwealth Avenue, Boston, Massachusetts. Their subject is "Leverage Reduction in Fractures of the Surgical Neck of the Humerus." Page 924.

HAWES, JOHN B. 2ND. A.B., M.D. Harvard University Medical School 1903. President, Boston Tuberculosis Association. Director, Rutland Cottage Sanatoria. Director, Massachusetts Tuberculosis League and National Tuberculosis Association. Address: 11 Marlborough Street, Boston, Massachusetts. Associated with him is

STONE, MOSES J. M.D. Tufts College Medical School 1921. Assistant Professor in Diseases of the Chest, Boston University School of Medicine. Examiner in the Tuberculosis Clinics of the Boston Health Department. Physician at the Chest Clinic of the Massachusetts Memorial Hospitals. Assistant in Medicine, Beth Israel Hospital. Address: 11 Marlborough Street, Boston, Massachusetts. Their subject is "Progress in Tuberculosis." Page 925.

MISCELLANY

REPORT OF THE COMMITTEE IN CHARGE OF THE EMERGENCY CAMPAIGN OF 1934

OCTOBER 25, 1934

Gross subscriptions to the Campaign, including donations made direct to agencies	$3,139,816.51
Cancellation of campaign subscriptions to date	7,177.39
Net subscriptions	$3,132,639.12
Amount receivable on pledges, October 25, 1934	$ 106,923.15

Cancellation of pledges to date, two-tenths of 1%.

All funds received have been deposited in The First National Bank of Boston, Treasurer. The accounting department has been under the direction of Messrs. Lybrand, Ross Bros. & Montgomery. Distributions to the participating agencies have been made monthly in proportion to their quotas. Donations designated for a particular agency have, of course, gone wholly to that agency.

Of the net subscriptions of $3,132,639.12, the participating agencies have received *through September 30*, a total of $2,128,110.57, distributed as follows:

For Relief — Family Service, Single Men and Women, Homes for Aged	$784,099.70
Hospitals, Dispensaries, Clinics, Care of Sick in Homes and Health Education	743,526.06
Settlement Houses and morale sustaining organizations	409,244.97
Care, Protection and Guidance of Children	176,524.84
Other organizations whose activities are not classified above	14,715.00

The Citizens' Committee, after careful study, has decided to conduct another campaign in 1935. The Committee is convinced that such a joint appeal in 1935 is essential to maintain the work of the hospitals, private charities and social agencies which participated in the 1934 Campaign. Although the government is spending huge sums on relief, the government is not assuming and cannot assume the more varied and more difficult services rendered in good times and bad by these private institutions.

The Citizens' Committee has chosen Mr. Oscar W. Haussermann as General Chairman of the 1935 drive. May I ask for him your coöperation and support?

J. W. FARLEY, *General Chairman.*

CORRESPONDENCE

AN OPEN LETTER TO DR. SHALER RICHARDSON

November 3, 1934.

Dr. Shaler Richardson,
Secretary-Treasurer-Editor,
Florida Medical Association, Inc.,
111 West Adams Street,
Jacksonville, Florida.

My dear Dr. Richardson:

My attention has been called to that portion of the issue of the *New England Journal of Medicine*, of October 11, 1934, which contains a letter written by you to the Managing Editor of the *New England Journal of Medicine.*

You write that "no action has been taken by the Florida Medical Association as an organization," and then you express your "belief" with regard to the "opinion of the members of the Association".

You sign the letter not as an individual, but as an officer of the Florida Medical Association, Inc., for you recite "Secretary-Treasurer-Editor, Florida Medical Association, Inc." In other words, you purport in your letter to be writing as an individual, but end up by writing in the capacity of an official, thereby conveying the impression that the organization desired to go on record unofficially, that the Medical Society of the County of New York was justified in its action.

You have expressed your "belief". I do not know whether you took the trouble to ascertain the at-

titude of at least two County Medical Societies, which are constituent parts of your Medical Association. I call your attention to the fact, if you do not know it already, that the Sarasota County Medical Society on November 21, 1933, unanimously passed a resolution expressing its confidence in "the Professional honesty and integrity of Dr. Fred Albee," and they "absolve him from any responsibility" relating to the matter concerning which you express your "belief".

I also call your attention, if you do not know it at this time, to the resolution of the Manatee County Medical Society, unanimously passed, wherein it stated "we have found Dr. Albee's standard of conduct to be beyond reproach," and that by means of the resolution they desired to express their "continued faith in Dr. Albee". Further, I wish to say that the resolutions of both these County Societies were passed after the New York County Society brought its charges.

I also desire to call your attention to the fact that the action of the Medical Society of the County of New York was subjected to review by the Supreme Court of the State of New York, in a suit brought by me against it to annul its resolutions and to command it to reinstate me. After the trial the Supreme Court Justice said: "It is clear that the evidence completely exonerates the petitioner (Dr. Albee) of any knowledge or consent in the preparation, printing or distribution of the handbills and the menu. It was an error for the respondent (County Medical Society of New York) to suspend him." A final order will be entered within a few days commanding the Medical Society of the County of New York to reinstate me.

I have been exonerated and vindicated by a Court of Justice, which considered the matter impartially and fairly. It did not have any axe to grind.

I note that you do not deny that there is a spirit of jealousy on the part of some doctors practicing in Florida. I do not think you can deny it, for your voluntary letter speaks for itself.

Very truly yours,

FRED H. ALBEE, M.D.

57 West 57th Street, New York City.

RECENT DEATHS

McGILLICUDDY—RICHARD ALOYSIUS MCGILLICUDDY, M.D., of 113 Avenue A, Turners Falls, Mass., died November 4, 1934. He was born in Springfield, Mass., in 1878, and received his M.D. degree from the Baltimore Medical College in 1903. After settling in Turners Falls, he served as Associate Medical Examiner and a Member of the Montague School Board. He was school physician for many years and joined the Massachusetts Medical Society in 1904. His widow and four children survive him.

MARTIN — GEORGE FORREST MARTIN, M.D., of 45 Harvard Street, Lowell, Mass., died November 4, 1934, at his home. He was born at Mason, Ohio, in 1862, the son of John Douglas and Caroline T. (Thompson) Martin. After graduating from the Lowell High School he studied medicine at, and graduated from, the New York Homeopathic Medical College and Flower Hospital in 1890. He formerly practiced at Skaneateles, New York, and in 1893 moved to Lowell, Mass.

Dr. Martin was a member of the Massachusetts Homeopathic Medical Society (ex-President), the Massachusetts Medical Society, the American Medical Association, the American College of Surgeons and other professional groups. He was instrumental in the development of the Lowell General Hospital where he served on the surgical staff and as a trustee. He was especially active in public health work.

He was a 33d degree Mason and an important member of many civic organizations.

During the World War he was a member of the public safety commission and chairman of its hospital division.

Dr. Martin is survived by his widow, Mrs. Gertrude Darling (Hunter) Martin, and a daughter, Mrs. Marion Martin Teeson, of Southbridge.

NOTICES

The Massachusetts Medical Society

SECOND ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee:

Barnstable

Sunday, November 18, at 4:00 P.M., at the Cape Cod Hospital, Hyannis. Subject: Cardiovascular Disease (First Session). John I. B. Vail, M.D., Chairman.

Berkshire

Thursday, November 22, at 4:30 P.M., at the House of Mercy Hospital, Pittsfield. Subject: Surgery. Albert C. England, M.D., George S. Reynolds, M.D., Chairmen.

Bristol North (Taunton Section)

Wednesday, November 21, at 7:30 P.M., at the Morton Hospital, Taunton. Subject: Surgery (Third Session). Arthur R. Crandell, M.D., Chairman.

Bristol South (Fall River Section)

Monday, November 19, at 4:00 P.M., at the Stevens Clinic of the Union Hospital, Prospect Street, Fall River. Subject: Cardiovascular Disease (Third Session). Eugene A. McCarthy, M.D., Sub-Chairman.

Essex North

Tuesday, November 20, at 4:00 P.M., at the Hotel Bartlett, 95 Main Street, Haverhill. Subject: Cardiovascular Disease (Second Session). Francis W. Anthony, M.D., Chairman.

Essex South
Tuesday, November 20, at 4:00 P.M., at the Salem Hospital, Salem. Subject: Endocrinology (Second Session). Walter G. Phippen, M.D., Chairman.

Franklin
Wednesday, November 21, at 8:00 P.M., at the Franklin County Public Hospital, Greenfield. Subject: Surgery (Second Session). Halbert G. Stetson, M.D., Chairman.

Hampden
Thursday, November 22, at 4:00 P.M. in the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:00 P.M., at the Holyoke City Hospital, Holyoke. Subject: Surgery (Second Session). George L. Schadt, M.D., Chairman.

Hampshire
Wednesday, November 21, at 4:15 P.M., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Subject: Pediatrics (Third Session). Robert B. Brigham, M.D., Chairman.

Middlesex East
Wednesday, November 21, at 4:00 P.M., at the Melrose Hospital, Melrose. Subject: Surgery (First Session). Joseph H. Fay, M.D., Chairman.

Middlesex North
Friday, November 23, at 7:00 P.M., at the St. John's Hospital, Lowell. Subject: Obstetrics and Gynecology (First Session). Frederick P. Murphy, M.D., Chairman.

Middlesex South
Monday, November 19, at 4:30 P.M., at the Cambridge Hospital, Mt. Auburn Street, Cambridge. Subject: Obstetrics and Gynecology (Second Session). Charles E. Mongan, M.D., Chairman.

Norfolk
Monday, November 19, at 4:00 P.M., at the Faulkner Hospital, Jamaica Plain. Subject: Cardiovascular Disease (Third Session). Hugo B. C. Riemer, M.D., Chairman.
Friday, November 23, at 8:30 P.M., at the Norwood Hospital, Norwood. Subject: Obstetrics and Gynecology (First Session). Hugo B. C. Riemer, M.D., Chairman.

Norfolk South
Monday, November 19, at 8:30 P.M., at the Quincy City Hospital, Quincy. Subject: Pediatrics (Second Session). David L. Belding, M.D., Chairman.

Plymouth
Tuesday, November 20, at 4:00 P.M., at the Brockton Hospital, Brockton. Subject: Obstetrics and Gynecology (Third Session). Walter H. Pulsifer, M.D., Chairman.

Worcester
Wednesday, November 21, at 7:30 P.M., in the Auditorium of the Nurses' Home of the Memorial Hospital, Worcester. Subject: Surgery (Second Session). Erwin C. Miller, M.D., Chairman.

Worcester North (Ayer Section)
Thursday, November 22, at 8:00 P. M., at the Ayer Community Memorial Hospital, Ayer. Subject: Industrial Medicine (First Session). Frank S. Bukeley, M.D., Chairman.

Worcester North (Fitchburg Section)
Friday, November 23, at 4:30 P.M., at the Burbank Hospital, Fitchburg. Subject: Surgery (First Session). Edward A. Adams, M.D., Chairman.

AN ADDRESS BY DR. J. G. DUSSER de BARENNE

On Tuesday, November 20, at 2:00 P.M., Dr. J. G. Dusser de Barenne, Sterling Professor of Physiology in the Yale University School of Medicine, will speak on "Some Recent Studies in Laminar Corticology" at the Reception Building of the State Hospital for Mental Diseases, Howard, Rhode Island.

PROGRAM ADOPTED BY THE EXECUTIVE COMMITTEE OF THE BOSTON HEALTH LEAGUE

1934-1935*

I. Continuing Committees:

A. Cancer
1. Publication of *Cancer Bulletin.*
2. Lectures: Medical lecture for adult groups. Lectures on biology of cancer for high school and college students.
3. Study of one hundred consecutive admissions to each cancer clinic in Boston to determine residence of patient, ability to pay and final disposition.
4. Distribution of slips giving Danger Signals of Cancer and listing Boston Clinics through the Metropolitan Life Insurance Company and the John Hancock, and to the employees of Boston department stores.

B. Child Health
1. Revision of card for physical examination; presenting the matter to the child caring agencies.
2. Continuance of interest in summer camp standards.

C. Health Education
1. Series of discussions planned for winter concerning programs of official and voluntary health agencies in order to advance coöperative efforts in health education.

*This program is subject to modification should new situations make changes desirable.

2. All members of the Health League are earnestly requested to lay before this Committee any health education project in the promotion of which the Committee can assist.

D. Infant and Maternal Mortality
1. Refer to this committee for further study material collected during 1933 in Charlestown and the West End.
2. Consideration of the advisability of publishing specific data on maternal mortality in Boston, since the rate is unduly high in this city.

E. Needs of Ward 9
Planning a coöperative exhibition on nutrition.

F. Pneumonia
1. Further study of results for 1934 and continuation of effort to reduce mortality.
2. A special study of all cases nursed by the Community Health Association, special article for the *New England Journal of Medicine*, and distribution of reprints to practicing physicians of Boston through their county medical societies is planned for this effort.

G. Social Hygiene
1. Sponsoring of the Staff Council on Syphilis and Gonorrhea provided its program justifies the continuance.
2. Follow up any current leads for furthering educational work on various social hygiene problems in coöperation with other League committees.
3. Coöperation with the Massachusetts Society for Social Hygiene in the Boston aspects of a study of social hygiene instruction in the educational system.
4. Sponsor the consultation service of the Massachusetts Society for Social Hygiene.
5. Check on social hygiene literature available in Boston libraries.

Committees With No Formal Program:

Chronic Disease, Dental, Domiciliary Medical Care, Mental Hygiene, Nursing, Tuberculosis.

II. Functional Activities:
Special committee to be appointed on Health Progress, whose duties will consist in dealing with recommendations made in special studies relating to health services in the City.

III. Office Activities:
A. Information Service
Directory of Clinical Facilities.
Directory of Hospital Facilities.
Directory of Summer Camps.
Monthly Calendar of Health Lectures and Radio Talks.

B. Tabulation of Health Statistics
Communicable Disease.
Infant Mortality.

C. Assistance to Committees.

D. Coöperate with other agencies by supplying health data when requested and by joint sponsoring of meetings.

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, November 22, in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May*.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

*No clinic scheduled for November 29 (Thanksgiving Day).

PETER BENT BRIGHAM HOSPITAL

GRADUATION EXERCISES, SCHOOL OF NURSING, NOVEMBER 23, 1934, ASSEMBLY HALL, SIMMONS COLLEGE

PROGRAM

Charles P. Curtis, President, Presiding

Address: Sally Johnson, R.N., B.S., Principal, Training School for Nurses, Massachusetts General Hospital, Boston.

Report of School: Carrie M. Hall, R.N., Principal, School of Nursing.

Announcement of Graduates: Mr. Curtis.

Presentation of Honorary Diploma of School of Nursing to Miss Johnson: Mr. Curtis.

Response: Miss Johnson.

Violin Solo: Wilhelmina Poor, 1936.

Awarding of Dr. John P. Reynolds Gold Medal.

Awarding of John P. Reynolds Scholarship: Dr. Joseph B. Howland, Superintendent of Hospital.

Presentation of Portrait of Carrie M. Hall to School of Nursing: Elsie Sayles Chase, 1921, President, Alumnae Association of School of Nursing.

Acceptance of Portrait: Mr. William Amory, Chairman of Advisory Committee to School of Nursing.

Response: Miss Hall.

March: School.

AN INVITATION TO FELLOWS OF THE MASSACHUSETTS MEDICAL SOCIETY

HARVARD UNIVERSITY MEDICAL SCHOOL COURSES FOR GRADUATES

November 7, 1934.

To the Editor of the *New England Journal of Medicine:*

I am sending you a list of activities in the Department of Pediatrics of the Children's Hospital and of

the Massachusetts General Hospital, to which members of the Massachusetts Medical Society are cordially invited. These exercises are offered without fees as a part of the Courses for Graduates, of the Harvard Medical School, to those who are interested in keeping in touch with Clinical Pediatrics, without enrolling in the prescribed courses.

The Children's Hospital and the Infant's Hospital:

Clinical-Pathological Conference — Thursdays, 12:00 M. (Amphitheatre).

Clinic—Medical, Surgical and Orthopedic Services—The first Monday in each month, 4:00 P.M. (Amphitheatre).

Clinic — Alternating Rounds between Surgical Service, Peter Bent Brigham Hospital (Amphitheatre) and Surgical and Orthopedic Services, Children's Hospital (Amphitheatre) —Thursdays, 4:30 P.M.

The Massachusetts General Hospital—The Children's Medical Service:

Clinical meeting of the Staff—Alternate Fridays, 12:00-1:00 P.M. (Ether Dome).

Ward Visit — Tuesdays, 2:30-4:00 P.M. (Massachusetts Eye and Ear Infirmary).

Seminar for discussion of recent investigations and literature — Tuesdays, 4:00-5:00 P.M. (Pediatric Laboratory).

MAYNARD LADD, M.D.,
In charge of Courses for Graduates,
Department of Pediatrics.

NOTICES OF MEETINGS

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance) Tuesday evening, November 27, at 8:15 P.M.

PROGRAM

Presentation of Cases.

Oscillometry in Medicine. By Dr. Alfred Friedlander, Dean, University of Cincinnati, College of Medicine.

Recent Advances in the Treatment of Obliterative Arterial Diseases of the Extremities. By Dr. Louis G. Herrmann, Assistant Professor of Surgery, University of Cincinnati, College of Medicine.

MARSHALL N. FULTON, M.D., *Secretary.*

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held in the Amphitheatre of the Mallory Institute of Pathology of the Boston City Hospital (Entrance on Massachusetts Avenue near Albany Street) Monday, November 26, at 8:15 P.M.

All members of the New England Heart Association and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary.*

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

The November meeting of the New England Ophthalmological Society will have a three-day program. It is to be held in conjunction with the annual meeting of the alumni of the Massachusetts Eye and Ear Infirmary. The regular meeting of the Society will be held on Tuesday, November 20, 1934 at the Infirmary, 243 Charles street, Boston. It will be followed on Wednesday by an all-day program arranged for the alumni. Thursday will be devoted to a visit at the American Optical Company, Southbridge, Mass. You are cordially invited to attend the entire three-day program.

PROGRAM

Tuesday, November 20:

9:00 A.M. Clinic and operating room.

11:30 A.M. Neuro-ophthalmological conference.

2:00 P.M. Ward rounds and clinico-pathological conference.

3:00 P.M. Analysis of fifty-five cases of toxic amblyopia, Dr. Frank D. Carroll.

3:30 P.M. Malnutrition in relation to ophthalmia neonatorum, Dr. Charles Walker.

4:00 P.M. Uveo-parotid disease, Dr. David Cogan.

4:30 P.M. Analysis of cataract operations, Dr. Herman Grossman.

5:00 P.M. Exhibition of new instruments and lighting apparatus, American Optical Company, Bausch & Lomb Optical Company.

6:00 P.M. Luncheon at the Massachusetts Eye and Ear Infirmary.

Evening program, 8:00 o'clock.

Muscle cases, Dr. Allen Greenwood.

Cases with hereditary eye conditions.

Paper: Heredity in Ophthalmology, Dr. Clyde E. Keeler, Howe Laboratory for Ophthalmic Research. Discussion: Professor Gregory Pincus, Institute of Biology, Harvard University.

BENJAMIN SACHS, M.D., *Secretary.*

NEW ENGLAND PHYSICAL THERAPY SOCIETY

NOTICE OF MEETING AND INVITATION TO THE PROFESSION TO ATTEND A SERIES OF LECTURES ON THE SUBJECT OF ELECTRICITY BY DR. L. L. CAMPBELL

The regular meeting of the New England Physical Therapy Society will be held at the Evans Memorial Auditorium, 82 East Concord Street, Boston, at 8 o'clock on Wednesday evening, November 21, 1934.

The program will be the first in a series of lectures on Electricity to be given by Dr. L. L. Campbell, Professor of Physics, Simmons College.

PROGRAM

Electricity: Dr. L. L. Campbell, Boston.

Lecture I:

Nature of Electricity.

Kinds of Electric Currents.

Apparatus for Production of Electric Currents.

Instruments for Measuring Electric Currents.

Electric Units.

The Electric Nature of Matter; Isotopes.

The lectures will include the topics of thermal, chemical and electromagnetic effects produced by electric currents and the physics of some electrotherapy apparatus. It will be of great value to physicians interested in the use of electrical currents in medical practice.

There will be an opportunity for general discussion.

All members of the medical profession are cordially invited to attend.

ARTHUR H. RING, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

November 15—Clover Hill Hospital will hold its next medical meeting at the hospital, 161 Berkeley Street, Lawrence, at 9 P.M.

November 15—Massachusetts General Hospital. Clinical Meeting will be held in the Moseley Memorial Building, 8:15-10 P.M.

November 15 and 22—Harvard Medical School. Lectures on "The Care of the Patient." Amphitheater C at 5 P.M. Thursday, November 15—Dr. Joseph H. Pratt of Boston. Thursday, November 22—Dr. Stephen Rushmore of Boston.

November 16—New England Roentgen Ray Society will meet at the Boston Medical Library, Boston, Mass., Friday, November 16, 1934, at 8:15 P.M. Scientific Session: "Diaphragmatic Hernia and Associated Conditions," Dr. Philemon E. Truesdale, Dr. Joseph H. Marks.

November 19—Boston Medical History Club will meet at the Boston Medical Library at 8 P.M. Pageant on the "Circulation and Respiration" given by students of Tufts College Medical School, under the direction of Dr. B. Spector. Everyone is invited to attend.

November 20—South End Medical Club will meet at 554 Columbus Avenue, Boston, at 12 noon.

November 20, 21, 22—New England Ophthalmological Society. See page 947.

MASSACHUSETTS DIETETIC ASSOCIATION

November 20—Tuesday, 2 P.M. Field Trip: Visit Kitchens of the Hotel Statler.

December 11—Tuesday, 8 P.M. "Food Allergy," Dr. Frank N. Allan, Department of Medicine, Lahey Clinic.

January 8—Tuesday, 8 P.M. "Careers and Natural Abilities," Professor Johnson O'Connor, Human Engineering Laboratory, Stevens Institute of Technology, Hoboken, N. J.

February 12—Tuesday, 8 P.M. "Diabetic Children," Dr. Priscilla White, Joslin Diabetic Unit.

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

November 21—New England Physical Therapy Society. See page 947.

November 22—Clinic at the Peter Bent Brigham Hospital. See page 946.

November 26—New England Heart Association. See page 947.

November 27—Harvard Medical Society. See page 947.

December 3-7—The Radiological Society of North America. See notice on page 562, issue of September 20.

December 6-8—Annual Conference of the National Society for the Prevention of Blindness. See page 736, issue of October 18.

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Semi-Annual Meeting will be held in January and the Annual Meeting in May, time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of January, March, and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

January 9, 1935—Melrose.
March 13, 1935—Wakefield.
May 8, 1935—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January—Goddard Hospital.
March—Plymouth County Hospital.
April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

November 28—Meeting at the Boston Medical Library. Speakers: Dr. Frederick T. Lord, Dr. Edward D. Churchill, Dr. Roderick Heffron. Subject: Symposium on Pneumonia.

January 23, 1935—General Meeting in association with the Boston Medical Library. Speaker and subject to be announced later.

March 27, 1935—Clinical Meeting at the Boston Lying-In Hospital.

April 24, 1935—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend all of these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.
HENRY T. HUTCHINS, M.D., Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

December 12—Wednesday evening. St. Vincent Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

January 9, 1935—Wednesday evening. Worcester City Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

February 13, 1935—Wednesday evening. Worcester State Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

March 13, 1935—Wednesday evening. The Memorial Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

April 10, 1935—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8, 1935—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.